Health Consultation

Assessment of Environmental Concerns and
Evaluation of Cancer Incidence in New Bedford's South End 1982-1998

RODNEY METALS
(a/k/a ALLEGHENY RODNEY STRIP DIVISION)
EPA FACILITY ID: MAD001067941

AND

BRITTANY DYEING AND PRINTING CORPORATION
EPA FACILITY ID: MAD001014612

NEW BEDFORD, BRISTOL COUNTY, MASSACHUSETTS

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Division of Health Assessment and Consultation
Atlanta, Georgia 30333

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Prepared by:

Massachusetts Department of Public Health
Bureau of Environmental Health Assessment
Community Assessment Program
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

Assessment of Environmental Concerns:
Rodney Metals and Brittany Dyeing and Printing Corporation and
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I. BACKGROUND AND STATEMENT OF ISSUES

At the request of concerned residents, Representative Antonio Cabral, and the New Bedford Health Department, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health Assessment (BEHA) conducted an evaluation of cancer incidence in the South End area of New Bedford, Massachusetts. Specifically, this evaluation was initiated based on community concerns about the possible role that environmental exposures related to the Allegheny Rodney Company (also known as Rodney Metals) and Brittany Dyeing and Printing Corporation (a.k.a. Brittany Dye) may play in the incidence of cancer in the neighborhoods adjacent to the facilities. Rodney Metals and Brittany Dye are located in the South End area of New Bedford, census tract 6528, on East Rodney French Boulevard to the south of Butler Street and east of Swan Street. Refer to Figure 1 for the location of the two facilities and the adjacent neighborhood.

II. OBJECTIVES

This investigation provides a review of potential exposure pathways to chemicals from Rodney Metals and Brittany Dye as well as a review of the pattern of cancer in the South End area of New Bedford through comparison of the incidence of eight cancer types with the incidence of these cancers in the state of Massachusetts as a whole. Additionally, available information about risk factors, including environmental factors, related to the development of cancer was evaluated. To evaluate concerns about potential environmental exposures from Rodney Metals and Brittany Dye, MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) to obtain and review available environmental data for these companies.

This report provides a descriptive evaluation of the occurrence of cancer in census tract 6528 in the city of New Bedford during the years 1982–1998, the time period for which the most recent and complete cancer incidence data were available from the Massachusetts Cancer Registry (MCR) at the initiation of this analysis. The city of New Bedford is divided into 30 smaller geographic areas or census tracts (CTs). The South End area of New Bedford as well as Rodney Metals and Brittany Dye are located in CT 6528, on the southern peninsula of the city. New Bedford CT 6528 comprises an area of approximately 1.8 square kilometers and has a total

population of 3,405 (U.S. DOC 2000). The location and boundaries of CT 6528 are also shown in Figure 1. A census tract is a smaller geographic subdivision of a city or town designated by the U.S. Census Bureau. Because age group and gender specific population information is necessary to calculate incidence rates, the census tract is the smallest geographic area for which cancer rates can be accurately calculated.

The results of this descriptive analysis can be useful in identifying cancer patterns or trends in a geographic context, to determine if a common cause or etiology is possible, and can serve to identify areas where further public health investigations or actions may be warranted. Descriptive analyses may also indicate that an excess of known risk factors associated with a disease, such as environmental exposures, exists in a certain geographic area. This descriptive analysis of cancer incidence data cannot be used to establish a causal link between a particular risk factor (either environmental or non-environmental) and the development of cancer. In addition, this analysis cannot determine the cause of any one individual's cancer diagnosis. The purpose of this evaluation is to report the findings on the patterns of cancer in the South End area of New Bedford and discuss them in the context of the available environmental information to determine whether recommendations for further public health action are needed. The specific objectives of this investigation were as follows:

- To evaluate opportunities for environmental exposure(s) to nearby residents to chemicals from Rodney Metals and Brittany Dye;

- To evaluate the incidence of eight cancer types (cancers of the bladder, breast, kidney, liver, lung, pancreas, leukemia, and non-Hodgkin's lymphoma) in New Bedford CT 6528 and specifically neighborhoods near Rodney Metals and Brittany Dye to determine if cancer is occurring more or less often than expected;

- To evaluate the geographic distribution of cancer in CT 6528 by mapping individual cases to determine whether any indication of an atypical pattern of cases exists in this area of the city, particularly in relation to possible exposure opportunities to chemicals from Rodney Metals or Brittany Dye;

- To review available descriptive information from the Massachusetts Cancer Registry (MCR) related to non-environmental risk factors for individuals diagnosed with cancer in New Bedford CT 6528; and

- To discuss the results of this evaluation in the context of the available scientific and medical literature on the eight types of cancer evaluated to determine whether further investigation or public health action is warranted.

III. COMMUNITY ENVIRONMENTAL CONCERNS

In order to address community environmental concerns, the MDEP Southeast Regional Office was contacted to obtain and review available environmental information pertaining to Rodney Metals and Brittany Dye. In addition to reviewing environmental data for the two facilities, information regarding other potential environmental sources located in the South End area of New Bedford and listed with MDEP as a location of a hazardous release or spill was reviewed (MDEP 2003a).

A. Rodney Metals

Rodney Metals was originally formed as Modern Venetian Blinds, which, in 1941, moved to 1357 East Rodney French Boulevard, on the east side of the peninsula of land separating Clarks Cove and New Bedford Harbor. The facility originally produced wooden slatted venetian blinds and over time introduced steel and aluminum to the product. Through this work, the company started rolling and selling strip and by the late 1950s, the venetian blind end of the business dwindled while the metal producing division expanded. In 1961, Rodney Metals was incorporated and continued to operate the Consumer Products Division, which housed the venetian blind part of the business. Teledyne, Inc. purchased Rodney Metals in 1967. In late 1999, the Rodney Metals Division became Allegheny Rodney. Today, operations consist of rolling steel in mills to various thicknesses, cutting the rolled steel to different lengths and widths, and coating the steel with mineral oil (Innovative Engineering Solutions, Inc. 2002).

The Rodney Metals facility is situated on one parcel of land approximately 8 acres in area and consists of a two-story building with a concrete floor. Landscaping and pavement compose the

remaining portions of the property. To the west, the facility is abutted by residential properties along Milbury Street. To the north is a parking lot located on Butler Street and currently used by employees of Rodney Metals. To the east, across East Rodney French Boulevard, are thin strips of undeveloped land and New Bedford Harbor. Brittany Dye abuts the Rodney Metals property to the south.

Rodney Metals first became air quality permitted by MDEP in 1987. The permit requires the facility to meet specific emission limits for particular pollutants. MDEP reviews permit applications and issues air quality permits based on emission control technologies and the standards set by the U.S. Environmental Protection Agency (EPA) for criteria pollutants such as nitrogen oxides (NO_x), sulfur oxides (SO_x), carbon monoxide (CO), particulates, and volatile organic compounds (VOCs). As established under the permit, stack emissions testing can be required at the time the permit is granted and/or if there is an opacity violation (e.g. a measurement of air quality), odor complaint, or other problem indicating the need for testing. According to MDEP Bureau of Waste Prevention, the Rodney Metals facility has received several notices of non-compliance in the past and has made adjustments as necessary to comply with their permit, including stack testing (MDEP 2003b).

In addition to air emissions, Rodney Metals has experienced some milling/mineral oil releases and at least one release of chlorinated VOCs resulting in contamination of soil and groundwater beneath the surface of the property and contamination of surface water of New Bedford Harbor. Evidence of subsurface oil contamination was first discovered during excavation activities for a new mill within the Rodney Metals building conducted in June 1994. The excavation site, located on the north side of the building, is approximately 60 feet from the parking lot to the north and approximately 200 feet from New Bedford Harbor to the east (Earth Tech 1995). Site assessments and remedial measures, including collection of groundwater and oil samples, revealed that the extent of the contamination was generally limited to the area beneath the sidewalk adjacent to the northern portion of the Rodney Metals facility. A passive oil recovery trench with an impermeable barrier was placed on the down-gradient side of the excavation trench to restrict oil and/or any other contaminants possibly present in oil from migration away from the release area (Innovative Engineering Solutions, Inc. 2000, 2002).

In 1997, during site assessment and remedial activities, VOCs were detected in groundwater from a monitoring well located up-gradient from the impermeable barrier and from a location adjacent to the building and across East Rodney French Boulevard. The source of the VOCs is suspected to be from a release of 66 gallons of 1,1,1-trichloroethane (TCA) that occurred near the former solvent storage area in October 1989 and migrated through groundwater (Innovative Engineering Solutions, Inc. 2002). In November 2001, during construction of a new drainage system in another area of the facility, another mineral oil release was discovered. Investigations of subsurface soils and groundwater indicated that the oil did not appear to be migrating significantly from the area (Innovative Engineering Solutions, Inc. 2002).

B. Brittany Dye

The Brittany Dye facility is located at 1357 East Rodney French Boulevard directly south of the Rodney Metals property on the New Bedford peninsula. Brittany Dye has operated for more than 100 years in the fabric dying and coating business and operates on an international scale (MDEP 2003b). In addition to Rodney Metals and residential areas to the north, Brittany Dye is abutted by New Bedford Harbor to the east, residential areas to the west, and a parking lot and undeveloped land directly to the south.

Brittany Dye has multiple emission stacks, and the facility is often in operation 24 hours a day. Four releases or spills of hazardous materials on the premises of Brittany Dye have been reported to MDEP (MDEP 2003a). These spills either occurred or were reported to MDEP between 1985 and 1991. One spill involved an unrecorded amount of dye from a drum; another involved an unknown amount of dye wastewater from a pipe or hose on the premises. Available information did not characterize the other two spills with respect to type of material released, amount of material released, or source of release. No environmental sampling data associated with the four releases could be located.

Brittany Dye is permitted by MDEP for air stack emissions and has received notices of non-compliance from MDEP periodically since the first air quality permit was issued in 1988. Similar to Rodney Metals, the air emissions permit granted to Brittany Dye sets limits for specific air emissions associated with their operations based on emission control technologies

and Massachusetts' compliance status with ambient air quality standards set by EPA such as NO_x, SO_x, CO, VOCs, and particulates. Air emissions testing at the facility can be required at the time the permit is issued by MDEP, and/or at subsequent times should opacity violations, odor complaints, and/or other problems associated with their operations occur (MDEP 2003b).

C. MDEP 21E Hazardous Material and Oil Releases in the New Bedford South End Area

In 1983, the Massachusetts Legislature established a statewide hazardous waste site cleanup program (the state Superfund program) under Chapter 21E of Massachusetts General Laws (M.G.L c21E, 310 CRM 40.0000). Under this legislation, the Massachusetts Department of Environmental Protection (MDEP) administers investigation and cleanup of hazardous material and oil release sites, known as "21E sites," in the Commonwealth.

The 21E sites are characterized by one or more releases of oil or other hazardous material. Releases can result from a variety of sources, including trucks/vehicles, underground storage tanks, and aboveground storage drums. Releases vary widely with respect to materials involved, the relative amount of materials released, and the geographic extent of contamination. Information on hazardous material and oil releases, including assessment and remedial response measures is available from 1977 – present from the MDEP Bureau of Waste Site Cleanup (MDEP 2003a).

MDPH reviewed the most recent information regarding hazardous material and/or releases located in the South End of New Bedford, CT 6528, and mapped the approximate location of sites with sufficient address information using a geographic information system (ESRI 2002). According to the most current information, there are several sites in CT 6528 characterized by more than one release (Figure 2). These sites include Rodney Metals (four releases, two of which were discussed previously), Brittany Dye (four releases), Fort Rodman at the southern tip of the New Bedford peninsula (nine releases), the New Bedford Water Pollution Control Facility located within the geographic boundaries of Fort Rodman (six releases), and 127 West Rodney French Boulevard (three releases). Four other sites, each characterized by one release, are located along West Rodney French Boulevard. Information specific to each release is provided in Table 1.

IV. REVIEW OF ENVIRONMENTAL SAMPLING DATA

To address concerns about possible environmental exposures associated with the two facilities, MDPH reviewed information from several reports on file with MDEP as well as the Toxics Chemical Release Inventory (TRI) data available from the U.S. Environmental Protection Agency (USEPA). Available environmental sampling data were reviewed, and a screening evaluation was conducted to identify substances that are either not expected to result in adverse health effects or substances that need to be considered for further analysis to determine whether they may be of potential health concern. The screening analysis identifies maximum concentrations of contaminants detected in various types of environmental media (i.e., air, soil, water) and compares these concentrations to health-based comparison values established by the U.S. Agency for Toxic Substances and Disease Registry (ATSDR) (ATSDR 2002a, 2002b). For this analysis, the maximum concentration of a substance detected in a particular environmental medium was used to compare to the appropriate health-based comparison value in order to conduct a conservative screening evaluation. If an ATSDR comparison value was not available for a specific chemical, the maximum detected concentration of that chemical was compared to the appropriate Method 1 groundwater or soil clean-up standards developed by MDEP (MDEP 1999).

The ATSDR comparison values are specific concentrations of a chemical for air, soil, or water that are used by health assessors to identify environmental contaminants that require further evaluation. These comparison values are developed based on health guidelines and assumed exposure situations that represent conservative estimates of human exposure. Chemical concentrations detected in environmental media that are less than a comparison value are not likely to pose a health threat. However, chemical concentrations detected in environmental media above a comparison value do not necessarily indicate that a health threat is present. In order for a compound to impact one's health, it must not only be present in the environmental media, but one must also come in contact with the compound. Therefore, if a concentration of a chemical is greater than the appropriate comparison value, the potential for exposure to the chemical should be further evaluated to determine whether exposure is occurring and whether health effects might be possible as a result of that exposure. The factors related to exposure that are unique to the

specific situation under investigation need to be considered to determine if an adverse health effect from this chemical could occur.

Rodney Metals Subsurface Sampling History

As discussed, in June 1994, an initial release of oil was encountered during excavation for a new mill at the Rodney Metals facility. The excavation site, located on the north side of the facility, is approximately 60 feet from the parking lot to the north and approximately 200 feet from New Bedford Harbor to the east (Earth Tech 1995). During field investigation activities in May 1995 oil was detected in three groundwater monitoring wells (MW-101, MW-102, and MW-109) situated in the northeast corner of the Rodney Metals facility where the rolling operations are based.

In 1997, analysis of samples taken from the groundwater and the subsurface milling oil at Rodney Metals indicated the presence of several chlorinated VOCs, specifically 1,1,1-trichloroethane (1,1,1-TCA), trichloroethylene (TCE), and several breakdown products of these VOCs, including vinyl chloride, 1,1-dichloroethane (1,1-DCA), and 1,1-dicloroethene (1,1-DCE). As previously stated, the source of the chlorinated VOCs in groundwater was speculated to be from a release of 66 gallons of 1,1,1-TCA that occurred in October 1989 in the eastern portion of the facility. According to New Bedford Fire Department records, at the time of the 1989 release, 46 gallons of the 1,1,1-TCA were recovered, and the remaining material was contained with absorbent. These vapor-degreasing solvents have not been used at the facility since 1995, when vapor-degreasing operations were replaced by the use of aqueous cleaning solutions (Earth Tech 1997, Innovative Engineering Solutions, Inc. 2000, 2002). In addition, chlorinated VOCs were detected in off-site surface water samples taken from New Bedford Harbor.

Groundwater Monitoring Wells

A sample of oil was obtained from MW-101, the only monitoring well that contained separate phase product. This sample was analyzed for PAHs and VOCs (Innovative Engineering Solutions, Inc. 2002). Several PAHs, including benzo(a)pyrene, fluoranthene, fluorene, 2-methylnaphthalene, and naphthalene were detected at concentrations exceeding ATSDR

comparison values for drinking water. In addition, the laboratory results of the oil sample indicated the presence of two VOCs, toluene (which is a component of mineral oil) and tetrachloroethene (PCE). Toluene was detected at a concentration of 100,000 ppb, and PCE was detected at a concentration of 3,200 ppb. The concentrations of both these compounds in the oil sample exceeded ATSDR drinking water comparison values, federal and state Maximum Contaminant Levels (MCL) for drinking water, and MDEP's Method 1 GW-2 standards. Refer to Table 2. It is important to note that MW-101 is located upgradient of the passive oil recovery trench, and therefore, migration of these compounds in the oil beyond the trench is not likely.

Due to the presence of PAHs and VOCs in the oil sample collected from MW-101, groundwater samples from each of the installed monitoring wells were analyzed for the presence of these two classes of compounds. PAHs were not detected in any of the groundwater samples; however, several VOCs, including 1,1,1-TCA and TCE, were detected at concentrations exceeding ATSDR comparison values and state and federal standards for drinking water. It is important to note that New Bedford's drinking water comes from surface water sources, and groundwater near Rodney Metals is not a source of drinking water. Therefore, use of comparison values established based on a drinking water exposure scenario represents a conservative approach. In MW-107, located across East Rodney French Boulevard and generally downgradient from MW-101, analytical results indicated the maximum detected concentrations of 1,1-dichloroethene (1,1-DCE) (30,000 ppb), 1,1,1-TCA (200,000 ppb), and TCE (29,000 ppb). All three of these detected VOCs exceeded their representative drinking water comparison value. Other VOCs detected in groundwater that exceeded drinking water comparison values were methylene chloride (3,200 ppb) in MW-106 and vinyl chloride (7 ppb) in MW-201. In MW-103, chlorobenzene and 1,1-DCA were detected at maximum concentrations of 2 ppb and 17 ppb, respectively. The detected concentrations of these VOCs did not exceed health based comparison guidelines. Further, no contaminants were detected in either MW-104 (situated in the northwest corner of the site) or MW-202 (located across East Rodney French Boulevard near the edge of New Bedford Harbor just northeast of the Rodney Metals building).

Subsurface Soil

During investigation activities in 1994, oil in soil located more than 3 feet below ground surface encountered during the construction of the mill within the Rodney Metals facility, was collected and analyzed. The results of the analysis indicated that Total Petroleum Hydrocarbons (TPH) was present at concentrations in subsurface soil ranging from 4,700 ppm (parts per million) to 34,000 ppm. Analytical results also indicated the presence of two metals, chromium (total) and lead. The maximum concentration of chromium detected was 6.3 ppm and lead was 5 ppm. Neither of these metals was detected at concentrations that exceeded their representative health-based comparison values, and both were below background levels for soils in the northwestern U.S. (ATSDR 1993). Refer to Table 3.

Surface Water

Because the Rodney Metals facility sits across from New Bedford Harbor, surface water samples were collected at two separate locations adjacent to the site, at low and high tide. Specifically, samples were taken to evaluate whether migration of VOCs via groundwater had occurred and if so, whether VOCs on the surface water posed a potential imminent hazard. The analytical results of the surface water samples indicated that 1,1,1-TCA was detected at 12 ppb and TCE was detected in surface water at 2 ppb. Both VOCs were below drinking water comparison values. Use of drinking water comparison values represents a conservative approach because the surface water from this area is not a source of drinking water. In addition, these concentrations are well below ambient water quality criteria (of 31,200 ppb for 1,1,1-TCA and 2,000 ppb for TCE), indicating that the VOCs present in surface water of New Bedford Harbor are not posing an imminent hazard and are not likely posing an unacceptable risk for people who might contact them in the surface water. Refer to Table 4.

Underground Utilities

To determine whether underground utilities (i.e., gas lines, electrical lines, storm sewer, sanitary sewer, water lines) were acting as a preferential migration pathway for the oil release, access points, such as manholes and electrical vaults, for the underground utilities located along East Rodney French Boulevard, near the facility, were visually inspected for the presence of oil. No

oil or sheen was visible in the samples collected for qualitative analysis. In addition, using an oxygen/explosion meter and a photoinonization detector (PID), the air, within each electrical vault and sanitary sewer that was checked, was screened from the ground surface. No readings of contaminants above background or ambient conditions were detected (Innovative Engineering Solutions, Inc. 2002).

In 2000, the Department of Public Works for the city of New Bedford installed new water lines along East Rodney French Boulevard in the vicinity of the Rodney Metals facility. In order to install these new water lines, soils were excavated to depths up to 12 feet below ground surface. Prior to the excavation activities, Rodney Metals notified the Department of Public Works of the possibility of encountering milling oil and chlorinated VOC-affected soils. During the excavation activities, no chlorinated VOCs or oil were encountered, indicating that the underground utilities along East Rodney French Boulevard were not acting as preferential migration pathways for the releases at the Rodney Metals facility (Innovative Engineering Solutions, Inc. 2002).

Rodney Metals and Brittany Dye Air Emissions Information

There are no ambient air sampling data available for the areas in the immediate vicinity of Rodney Metals or Brittany Dye. Both facilities operate with air quality stack emissions permits from MDEP. The most recent stack test was performed at Rodney Metals in November 1997. Routine stack sampling is not required under the air permitting program, and follow-up sampling is only triggered when it is determined to be necessary to address opacity, odors or other air quality problems associated with the facilities. Emission limits for pollutants such as NO_x, SO_x, CO, VOCs, and particulates were determined at the time the permits were issued and were established based on cost and availability of emission controls at the time of review as well as Massachusetts' level of attainment with EPA ambient air quality standards. According to MDEP, the agency is not aware of any existing non-compliance issues at either Rodney Metals or Brittany Dye. All notices of non-compliance issued to either facility in the past have been addressed (MDEP 2003b).

However, because air emissions from Rodney Metals and Brittany Dye are one of the main environmental concerns expressed by residents in the area surrounding the two facilities, a review of the Toxic Chemical Release Inventory database (TRI) was conducted. The TRI is a surveillance system within EPA, which estimates the annual releases of toxic chemicals to the environment. The system evolved from the Emergency Planning and Community Right-to-Know Act (EPCRA) and requires businesses to report the locations and quantities of chemicals stored on-site to state and local agencies to help communities prepare to respond to potential chemical spills and emergency releases (USEPA 2003). Although TRI annual release estimates cannot be used to specifically evaluate whether individuals living near the two facilities are actually being exposed to air emissions, the information can be helpful when evaluating the pattern of cancer and the likelihood that environmental factors may have played a role in their development in this area of New Bedford.

Review of TRI data for Rodney Metals as far back as 1987 indicates that the facility has reported several stack and/or fugitive (non-stack) emissions to the air including hydrogen fluoride, methyl ethyl ketone, methyl isobutyl ketone, and nitric acid (USEPA 2003). The compound 1,1,1-TCA was reported as a fugitive emission at the facility from 1987 through 1995, but was not reported as an emission after 1995. This is likely attributed to the facility's implementation of an aqueous cleaning operation in replacement of 1,1,1-TCA usage reported during that year. Although not included in the TRI information reviewed, TCE was used at the Rodney Metals facility prior to 1982 (Innovative Engineering Solutions, Inc. 2002), and it is possible that this compound may have also been emitted to the air in that time period. According to the TRI data, Brittany Dye reported stack and/or fugitive emissions of 1,1,1-TCA from 1987 to 1994 and fugitive emissions of TCE from 1995 through 2000 (USEPA 2003).

V. EVALUATION OF POTENTIAL COMMUNITY EXPOSURE PATHWAYS

An evaluation of potential pathways of exposure was conducted to determine whether releases or activities at the Rodney Metals and Brittany Dye sites could have impacted residents in the South End area. It is important to note that chemical concentrations detected in the environment do not necessarily represent a health threat. In order for a compound to impact one's health, it must not

only be present in a certain environmental media (i.e., air, soil or water), but one must also come into contact with the compound via the contaminated media through ingestion, inhalation, or skin absorption. Therefore, the presence of contaminants at a site alone does not necessarily constitute exposure. The exposure pathway analysis is an evaluation of the environmental and human components that could lead to contact with contaminants in the environment. The pathway analysis consists of five elements: a source of contamination, transport through an environmental medium (e.g., air, soil, water), a point of exposure, a route of human exposure, and an exposed population.

Exposure to a chemical must first occur before any potential adverse health effects can result. Five conditions must be present for exposure to occur. First, there must be a source of that chemical. Second, an environmental medium must be contaminated by either the source or by chemicals transported away from the source. Third, there must be a location where a person can potentially contact the contaminated medium. Fourth, there must be a means by which the contaminated medium could enter a person's body, such as ingestion, inhalation, and dermal absorption. Finally, the chemical must actually reach the target organ susceptible to the toxic effects caused by that particular substance at a sufficient dose and for a sufficient exposure time for an adverse health effect to occur (ATSDR 1993).

A completed exposure pathway indicates that exposure to humans occurred in the past, is occurring in the present, or will occur in the future. A completed exposure pathway exists when all of the five elements are present. A potential exposure pathway exists when one or more of the five elements is missing or uncertain and indicates that exposure to a contaminant could have occurred in the past, could be occurring in the present, or could occur in the future. An exposure pathway can be eliminated if at least one of the five elements is missing and will not likely be present in the future. The discussion that follows incorporates only those exposure pathways that are important and relevant to this evaluation in New Bedford.

A. Exposure to Groundwater and Soil

For residents living adjacent to the Rodney Metals facility, it is unlikely that past, present, and future exposures to contaminated groundwater and subsurface soil resulting from historical releases at the property are occurring. Groundwater flow is away from residential areas, and no

contamination was detected in the upgradient monitoring well located closest to the neighborhood (MW-104). Since groundwater in the area is not being used as a source of drinking water, ingestion is not a possible route of exposure for residents living in this part of New Bedford. Also, because the oil and VOC-affected groundwater is located below the concrete floor of the Rodney Metals facility, dermal contact to the contaminated groundwater is not a likely source of exposure for residents living adjacent to or in the vicinity of the Rodney Metals facility. Like groundwater, it is not likely that residents living in neighborhoods adjacent to the Rodney Metals facility would contact oil and VOC-affected soil. Investigations at the site indicated that contaminated soil is confined beneath the surface underneath the concrete floor of the facility. Furthermore, the pavement in the area also acts as a protective barrier precluding human contact to oil and VOC-affected soil. Therefore, for individuals residing in the vicinity of Rodney Metals, potential exposures via contaminated groundwater and subsurface soil have been eliminated.

Past exposure to oil may have been possible for workers who directly participated in subsurface activities associated with the construction of the mill at the facility in 1994. Such activity would have allowed the opportunity for workers to come in contact with contaminants in subsurface groundwater or any volatile contaminants contained in subsurface groundwater that could have been released to ambient air. Therefore, inhalation and dermal contact are the principle routes of exposure by which workers at the facility may have been potentially exposed in the past to oil-contaminated groundwater. Since the completion of this excavation activity and the affected area is below the foundation for the mill, future exposure through direct contact with contaminated groundwater is not likely for employees at the Rodney Metals facility. Furthermore, vapor-degreasing solvents, such as 1,1,1-TCA and TCE, have not been used at the facility since 1995; therefore, future releases of these chemicals are not expected to occur. However, future exposures to oil and chlorinated VOCs in groundwater may still be possible for workers if future excavation actions are undertaken at the facility in this area.

Like groundwater, past exposure to subsurface soil through inhalation of dust and dermal contact may have been possible for Rodney Metal workers involved in subsurface activities related to excavation for a new mill at the site in 1994. In addition, future exposures via inhalation and dermal contact may be possible if remedial activities are conducted in this area of the Rodney

Metals facility in the future. However, while past and future exposures to subsurface soils by workers represent potential exposure pathways, it is important to note that both lead and chromium were detected below health-based comparison values in subsurface soils.

Past results of investigations conducted at the manholes along East Rodney French Boulevard indicated that underground utilities did not appear, at the time, to be acting as a preferential migration pathway for the releases away from the Rodney Metals property. Further, in 2000, when the New Bedford Department of Public Works was installing new water lines in East Rodney French Boulevard in the vicinity of the Rodney Metals facility, soils were excavated up to 12 feet below ground surface. It was noted at this time that neither milling oil nor chlorinated VOCs were encountered during excavation activities. Therefore, it is suggested that the underground utilities are not acting as a migration pathway for the releases at Rodney Metals, and any potential exposures pathways associated with the underground utilities may be eliminated.

B. Exposure to Indoor Air

Volatilization of groundwater contaminants is possible in areas where basement flooding and/or vapor infiltration occur(s) from an underlying groundwater plume. Although chlorinated VOC vapors have the potential to migrate via groundwater into indoor air, as do some constituents of oil, this route of exposure does not appear likely for residents in the nearby neighborhood since all residents abutting the Rodney Metals facility are upgradient of the contaminated portion of the facility. As discussed, the groundwater at the Rodney Metals property flows east-northeasterly of the facility towards New Bedford Harbor and away from residential areas, and if the contamination were to migrate away from the property through groundwater, it would travel toward New Bedford Harbor, and not to the west where some of the residential neighborhoods are situated. In addition, results of the analysis of groundwater samples collected from the northwest corner of the facility and closest to the adjacent neighborhood (MW 104) did not indicate the presence of VOCs, PAHs, or total petroleum hydrocarbons (TPH). Based on available information reported in Innovative Engineering Solutions (2002) and discussed earlier, it does not appear that vapors from the groundwater contamination are migrating through underground utility channels, either.

Past, present, and future exposures to indoor air contaminated with oil constituents or chlorinated VOCs via volatilization from groundwater are possible for workers at the Rodney Metals facility. Because there is evidence of groundwater contamination beneath the northern and eastern portion of the facility, vapor permeation may be a source of exposure for workers at the facility. However, facilities equipped with ventilation systems would help to reduce the potential for indoor air exposures as a result of vapor permeation. In addition, although chlorinated VOCs are no longer used at the facility, these compounds as well as their metabolites generally slowly degrade in groundwater (unlike their presence in air, where breakdown occurs rapidly). Therefore, it is possible that chlorinated VOCs could remain in the subsurface media, such as groundwater and soil, for an extended period of time. Further, over time, intermittent exposure to these compounds may occur if there were to be volatilization to indoor air, particularly in poorly ventilated areas.

C. Exposure to Surface Water

Past exposure to surface water contaminated with chlorinated VOCs may have occurred for individuals who had contact with New Bedford Harbor, located east of the Rodney Metals facility, at the time of the release. The limited surface water sampling that was undertaken in New Bedford Harbor indicated that the presence of two chlorinated VOCs, 1,1,1-TCA and TCE, were detected below drinking water guidelines. Therefore, at the levels detected in surface water, adverse health effects would not be anticipated to occur if the area was/is used as a recreational area. The dominant fate of VOCs released to surface water is volatilization to the air (predicted half-life for TCE is minutes to hours), and bioconcentration in fish and other edible aquatic biota is not thought to be a dominant or significant fate process (ATSDR 1997, Wu and Schaum 2000). Further, due to concerns about polychlorinated biphenyl compound (PCB) contamination in New Bedford Harbor, MDPH promulgated regulations to close this area to commercial fishing in September 1979 (MGL 105 CMR 260.00). In addition, information from past MDPH studies have found that the majority of the general public in the greater New Bedford area was not catching and eating fish from these areas (MDPH 1987).

D. Exposure to Ambient Air

Past, current, and future exposures to contaminants emitted from Rodney Metals and Brittany Dye are possible from the ambient air surrounding the sites. However, it should be noted that exposures from inhalation and, to a lesser extent, dermal contact to contaminants in outdoor air would be appreciably less than exposures experienced in indoor or confined areas. This is because dilution factors and environmental parameters, such as seasonal variation and wind direction, would, to a much greater extent, affect the presence and levels of contaminants in outdoor air compared to indoor air resulting in lower potential for exposure to occur from outdoor versus indoor air. Nevertheless, potentially impacted populations in this area of New Bedford would include workers on site at Rodney Metals and Brittany Dye, residents abutting the facilities, and visitors to residential homes in adjacent neighborhoods, particularly those residential neighborhoods that might be downwind from the facilities. However, with the exception of air sampling conducted within manhole covers and catch basin grates, no on- or off-site ambient air monitoring data are available to evaluate potential inhalation exposures associated with emissions at these two facilities. While stack emission limits are established for both facilities through MDEP permits, other than TRI data, there are little to no data available to evaluate ambient air levels of the emissions released from these facilities over time and no way to specifically evaluate potential impacts these emissions may be having to air quality in the surrounding area.

During warmer months, exposure opportunities via solvent evaporation and stack emissions may be greater for residents in the area because individuals are more likely to open windows and doors or use air conditioners permitting outdoor air to enter homes. However, the physiochemical properties attributed to VOCs complicate this potential inhalation exposure scenario. For example, during the warmer months, interactions with the atmosphere cause the VOCs in ambient air to be broken down, resulting in lower levels and less persistence of VOCs in air and minimizing exposure potential. Furthermore, due to the high vapor pressures of VOCs, these compounds are expected to primarily exist in the vapor phase of the atmosphere rather than attached to particulate matter present in ambient air and, thus, would not tend to settle, aggregate, or linger (Wu and Schaum 2000).

VI. ANALYSIS OF CANCER INCIDENCE IN NEW BEDFORD CT 6528

A. Methods for Analyzing Cancer Incidence

1. Case Identification/Definition

Cancer incidence data, reports of new cancer diagnoses, for the years 1982–1998 were obtained for the city of New Bedford from the MCR, a division of the Bureau of Health Statistics, Research and Evaluation within MDPH. Eight cancer types were evaluated in this investigation and include cancers of the bladder, breast, kidney, leukemia, liver, lung, non-Hodgkin's lymphoma, and pancreas. These cancer types were selected for evaluation based on elevations that were observed at the city level in a preliminary review of cancer rates in New Bedford and resident concern over suspected elevations in some of these cancer types in the South End area. Only cases reported to the MCR as a primary cancer for one of the eight cancer types and diagnosed among a resident of New Bedford CT 6528 were included in the analysis. Cases were selected for inclusion based on the address reported to the hospital or reporting medical facility at the time of diagnosis. Cases for which census tract designation was not possible were excluded from the analysis.

The MCR is a population based surveillance system that began collecting information on Massachusetts residents diagnosed with cancer in the state in 1982. All newly diagnosed cancer cases among Massachusetts residents are required by law to be reported to the MCR within six months of the date of diagnosis (M.G.L. c.111s.111B). The 17-year period 1982–1998 constitutes the period for which the most recent and complete cancer incidence data were available from the MCR at the time of this analysis.

The term "cancer" is used to describe a variety of diseases associated with abnormal cell and tissue growth. Epidemiologic studies have revealed that different types of cancer are individual diseases with separate causes, risk factors, characteristics, and patterns of survival (Berg 1996). Cancers are classified by the location in the body where the disease originated (the primary site) and the tissue or cell type of the cancer (histology). Therefore, each of the cancer types reviewed in this report was evaluated separately. Cancers that occur as the result of the metastasis or the

spread of a primary site cancer to another location in the body are not considered as separate cancers and therefore, were not included in this analysis.

It should be noted that the MCR research file might contain duplicate reports of individuals diagnosed with cancer. Duplicate cases are additional reports of the same primary site cancer case. In New Bedford CT 6528, no duplicate reports were identified during the years 1982–1998. However, reports of individuals with multiple primary site cancers were included as separate cases in the analyses in this report. A multiple primary cancer case is defined by the MCR as a new cancer in a different location in the body, or a new cancer of the same histology (cell type) as an earlier cancer, if diagnosed in the same primary site (original location in the body) more than two months after the initial diagnosis (MCR 1996).

2. Calculation of Standardized Incidence Ratios (SIRs)

To determine whether elevated numbers of cancer cases occurred in New Bedford CT 6528, the South End area, cancer incidence data were tabulated by gender according to six age groups to compare the observed number of cancer cases to the number that would be expected based on the statewide cancer rate. Standardized incidence ratios (SIRs) were then calculated for the period 1982–1998 for each of the eight primary cancer types for New Bedford CT 6528. SIRs were also calculated for three smaller time periods, 1982–1986, 1987–1994, and 1995–1998, in order to evaluate patterns or trends in cancer incidence over time.

In order to calculate SIRs, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980, 1990, and 2000 U.S. census data for New Bedford CT 6528 (U.S. DOC. 1980, 1990, 2000). Midpoint population estimates were calculated for each time period evaluated (i.e., 1984, 1990 and 1996). To estimate the population between census years, an assumption was made that the change in population occurred at a constant rate throughout the ten-year interval between each census.

3. Interpretation of a Standardized Incidence Ratio (SIR)

An SIR is an estimate of the occurrence of cancer in a population relative to what might be expected if the population had the same cancer experience as a larger comparison population designated as "normal" or average. Usually, the state as a whole is selected to be the comparison

population. Using the state of Massachusetts as a comparison population provides a stable population base for the calculation of incidence rates.

Specifically, an SIR is the ratio of the observed number of cancer cases in an area to the expected number of cases multiplied by 100. The population structure of each town is adjusted to the statewide incidence rate to calculate the number of expected cancer cases. The SIR is a comparison of the number of cases in the specific area (i.e., city, CT) to the statewide rate. Comparisons of SIRs between towns or census tracts are not possible because each community has different population characteristics.

An SIR of 100 indicates that the number of cancer cases observed in the population being evaluated is equal to the number of cancer cases expected in the comparison or "normal" population. An SIR greater than 100 indicates that more cancer cases occurred than were expected, and an SIR less than 100 indicates that fewer cancer cases occurred than were expected. Accordingly, an SIR of 150 is interpreted as 50% more cancer cases than the expected number; an SIR of 90 indicates 10% fewer cancer cases than expected.

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. For example, an SIR of 150 based on four expected cases and six observed cases indicates a 50% excess in cancer, but the excess is actually only two cases. Conversely, an SIR of 150 based on 400 expected cases and 600 observed cases represents the same 50% excess in cancer, but because the SIR is based upon a greater number of cases, the estimate is more stable. It is very unlikely that 200 excess cases of cancer would occur by chance alone. As a result of the instability of incidence rates based on small numbers of cases, SIRs were not calculated when fewer than five cases were observed for a particular cancer type.

4. Calculation of the 95% Confidence Interval

To help interpret or measure the stability of an SIR, the statistical significance of each SIR was assessed by calculating a 95% confidence interval (95% CI) to determine if the observed number of cases is "significantly different" from the expected number or if the difference may be due solely to chance (Rothman and Boice 1982). Specifically, a 95% CI is the range of estimated

SIR values that have a 95% probability of including the true SIR for the population. If the 95% CI range does not include the value 100, then the study population is significantly different from the comparison or "normal" population. "Significantly different" means there is less than a 5% chance that the observed difference (either increase or decrease) is the result of random fluctuation in the number of observed cancer cases.

For example, if a confidence interval does not include 100 and the interval is above 100 (e.g., 105–130), there is a statistically significant excess in the number of cancer cases. Similarly, if the confidence interval does not include 100 and the interval is below 100 (e.g., 45–96), the number of cancer cases is statistically significantly lower than expected. If the confidence interval range includes 100, the true SIR may be 100. In this case, it cannot be determined with certainty that the difference between the observed and expected number of cases reflects a real cancer increase or decrease or is the result of chance. It is important to note that statistical significance does not necessarily imply public health significance. Determination of statistical significance is just one tool used to interpret SIRs.

In addition to the range of the estimates contained in the confidence interval, the width of the confidence interval also reflects the stability of the SIR estimate. For example, a narrow confidence interval, such as 103–115, allows a fair level of certainty that the calculated SIR is close to the true SIR for the population. A wide interval, for instance 85–450, leaves considerable doubt about the true SIR, which could be much lower than or much higher than the calculated SIR. This would indicate an unstable statistic. Due to the instability of incidence rates based on small numbers of cases, statistical significance was not assessed when fewer than five cases were observed.

5. Evaluation of Cancer Risk Factor Information

Available information reported to the MCR related to risk factors for cancer development was reviewed and compared to known or established incidence patterns for the cancer types evaluated in this report. This information is collected for each individual at the time of cancer diagnosis and includes age at diagnosis, stage of disease, smoking status and occupation. One or even several factors acting over time can be related to the development of cancer. For example, tobacco use has been linked to lung, bladder, and kidney cancers. Other cancer risk factors may

include lack of crude fiber in the diet, high fat consumption, alcohol abuse, and reproductive history. Heredity, or family history, is an important factor for several cancers. To a lesser extent, some occupational exposures, such as jobs involving contact with asbestos, have been shown to be carcinogenic (cancer causing). Environmental contaminants have also been associated with certain types of cancer. The available risk factor information from the MCR was evaluated for individuals diagnosed with cancers that were elevated in New Bedford CT 6528. However, information about personal risk factors that might include family history, hormonal events, diet, and similar factors that may also influence the development of cancer is not collected by the MCR, and therefore, it was not possible to evaluate them in this investigation.

6. Determination of Geographic Distribution of Cancer Cases

In addition to calculation of SIRs, address at the time of diagnosis for each individual diagnosed with cancer was mapped using a computerized geographic information system (GIS) (ESRI 1999). This allowed assignment of census tract location for each case as well as an evaluation of the spatial distribution of individual cases at a smaller geographic level within a census tract (i.e., neighborhoods). The geographic pattern was determined using a qualitative evaluation of the point pattern of cases in New Bedford CT 6528. In instances where the address information from the MCR was incomplete, that is did not include specific streets or street numbers, efforts were made to research those cases using telephone books and city residential lists issued within two years of an individual's diagnosis. For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in this report. [Note: MDPH is bound by law not to reveal the name or identifying information of an individual diagnosed with cancer whose case is reported to the MCR.]

B. Results of Cancer Incidence Analysis

The following section presents cancer incidence rates for New Bedford CT 6528 during the 17-year time period 1982–1998. These data are summarized in Tables 3 through 6. To evaluate possible trends over time, these data were also analyzed by three smaller time periods, 1982–1986, 1987–1994, and 1995–1998. SIRs were not calculated for some cancer types in smaller time periods due to the small number of observed cases (less than five). However, the expected

number of cases was calculated during each time period, and the observed and expected number of cases was compared to determine whether excess numbers of cancer cases were occurring.

1. Cancer Incidence in New Bedford CT 6528

During the overall time period 1982–1998, cancer incidence in New Bedford CT 6528 occurred approximately at or near the expected rate for the eight cancer types evaluated. Some rates were higher or lower but generally, not at a level of statistical significance. The exceptions included lung cancer, which occurred statistically significantly less often than expected among males and females combined as well as among females, and leukemia, which was statistically significantly elevated in this area of New Bedford among females.

Bladder cancer occurred at approximately the expected rate in New Bedford CT 6528 during each time period evaluated and during the overall 17-year time period 1982–1998 (14 cases observed vs. 16 expected). During the most recent time period, 1995–1998, four individuals were diagnosed with bladder cancer versus 3.5 expected.

The incidence of breast cancer was about as expected during 1982–1998 (56 cases observed vs. 57.5 expected). One male was diagnosed with breast cancer during the 17-year period. During the earliest time period 1982–1986, 15 individuals were diagnosed with breast cancer compared to 14.8 expected. During 1987–1994, breast cancer occurred slightly less often than expected (24 cases observed vs. 27.9 expected). Finally, during the most recent time period, 1995–1998, more individuals in New Bedford CT 6528 were diagnosed with breast cancer than expected (17 cases observed vs. 13.7 expected, SIR=124). However, this elevation was based on approximately 3 additional cases over the expected number and was not statistically significant (95% CI =72–199).

The incidence of kidney cancer was greater than expected in CT 6528 based on the state rate for the overall time period 1982–1998 (12 cases observed vs. 8.7 expected, SIR=139). During the earlier time periods 1982–1986 and 1987–1994, kidney cancer occurred about as often as expected in CT 6528. The elevation observed in the overall time period 1982–1998 was primarily due to an increase in diagnoses among males in this census tract during the most recent time period 1995–1998 (5 cases observed vs. 1.3 expected, SIR=380, 95% CI=123–887).

Although this result was statistically significant, it was based on a relatively small number of cases and the wide 95% confidence interval indicate that the increased SIR is somewhat unstable. Therefore, it is uncertain, based on this data, whether the increase represents a true elevation in the incidence of kidney cancer among males in this area of New Bedford. Females experienced kidney cancer approximately at or near the rate expected during each time period.

Leukemia occurred more often than expected in New Bedford CT 6528 during the 17-year time period 1982–1998 (12 cases observed vs. 7 expected, SIR=171, 95% CI=88–299). This elevation was due to a statistically significant elevation in the incidence of this cancer type among females during 1982–1998 (8 cases observed vs. 2.9 expected, SIR=272, 95% CI=117–537). When leukemia incidence rates were evaluated over time, in general, males were diagnosed with leukemia at approximately the rate expected while females were diagnosed slightly more often than expected during each time period. However, these elevations were based on approximately one to two additional cases during each smaller time period, and the elevations were not statistically significant. Of note, no diagnoses of leukemia were reported among children in CT 6528 during the 17-year time period 1982–1998.

Residents of New Bedford CT 6528 experienced liver cancer at about the rate expected during 1982–1998 (2 cases observed vs. 2 expected) and during each smaller time period evaluated, 1982–1986, 1987–1994, and 1995–1998.

Overall, during the 17-year time period 1982–1998, the incidence of lung cancer was statistically significantly lower than expected among males and females combined in this area of the city (41 cases observed vs. 58.1 expected, SIR=71, 95% CI=51–96) and among females when evaluated separately by gender (10 cases observed vs. 22.2 expected, SIR=45, 95% CI=22–83). Although not statistically significant, males in New Bedford CT 6528 also experienced a lower than expected rate of lung cancer during this time period (31 cases observed vs. 36 expected, SIR=86). Similar trends were observed when incidence rates were evaluated for lung cancer over time.

Non-Hodgkin's lymphoma (NHL) occurred slightly less often than expected in New Bedford CT 6528 during 1982–1998 (10 cases observed vs. 12.9 expected, SIR=77). Further, this trend was consistent when males and females were evaluated separately (5 cases among males observed vs.

6.8 expected; 5 cases among females observed vs. 6.1 expected). During each of the smaller time periods evaluated, (1982–1986, 1987–1994, and 1995–1998), NHL occurred approximately at or below the expected rate. However, during 1987–1994, five females in CT 6528 were diagnosed with NHL where three cases were expected. This slight elevation was not statistically significant. Moreover, the incidence of NHL among females has declined in the most recent time period 1995–1998 (0 cases observed vs. 1.6 expected).

A slight elevation in the incidence of pancreatic cancer was observed in New Bedford CT 6528 during the 17-year time period 1982–1998 (10 cases observed vs. 8.4 expected). However, this slight elevation was based on about one additional case over the expected number and was not statistically significant. Similar rates were observed when males and females were evaluated separately by gender (5 cases observed among males vs. 4.1 expected; 5 cases observed among females vs. 4.2 expected). During 1982–1986 and 1987–1994, pancreatic cancer occurred at about the rates expected. However, during the most recent time period, 1995–1998, a slight elevation was observed where four individuals were diagnosed with pancreatic cancer and two cases were expected. The two additional cases were diagnosed among females (3 cases observed vs. 1 expected). Again, this slight elevation was based on an increase of two cases over the expected number and was not statistically significant.

2. Review of Cancer Risk Factor Information

As previously mentioned, cancer is not just one disease but is a term used to describe a variety of different diseases. As such, studies have generally shown that different cancer types have different causes, patterns of incidence, risk factors, latency periods (period between exposure and development of disease), characteristics, and trends in survival. Available information from the MCR related to age and gender, as well as other factors related to the development of cancer such as smoking and occupation, were reviewed for those cancer types that had statistically significant elevations in incidence in New Bedford CT 6528. These cancer types included leukemia and kidney cancer. Information for each of these cancer types was compared to known or established incidence trends to assess whether any unexpected patterns exist among these cases. For detailed information regarding risk factors associated with these and the other six cancer types evaluated in this report, please refer to Appendix A.

Age and gender are risk factors in many types of cancers, including leukemia and kidney cancer. A review of age-group specific SIRs by census tract was not possible because of the small numbers of cases in each group. However, where there was a statistically significant elevation of cancer cases in New Bedford CT 6528, the distribution of cases by age was reviewed.

Tobacco use is also a known or suggested causal risk factor in several types of cancer, including kidney cancer. The smoking status of individuals diagnosed with this cancer in New Bedford CT 6528 during the years 1982–1998 was reviewed. However, results of smoking status analysis should be interpreted with caution because of the number of individuals for which smoking status was unknown.

In some studies, an association has been found with exposures to specific occupations and an increase in incidence of kidney cancer and leukemia. Therefore, occupational information as reported by the MCR at the time of diagnosis was reviewed for individuals diagnosed with these two cancer types in CT 6528 to determine the role that occupational factors may have played in the development of these cancers in this area of New Bedford. It should be noted, however, that occupational data reported to the MCR are generally limited to job title and often do not include specific job duty information that could further define exposure potential for individual cases. Further, these data are often incomplete as occupational information can be reported as unknown, at home, or retired.

As described below, leukemia describes a group of different cancers that occur in the blood-forming organs and is classified by cell type. The different types of leukemia occur with different frequencies in the general population. Therefore, histologic (cell type) distribution was reviewed for diagnoses of leukemia in New Bedford CT 6528. Patterns of disease were compared to known or established incidence trends to assess whether any unusual patterns exist in these areas.

1. Leukemia

(a) Histology, Age, and Gender Distribution

During the period 1982–1998, a statistically significant elevation in the incidence of leukemia among females was noted in New Bedford CT 6528. Leukemia is classified into four main sub-

types: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also several rare types of leukemia (e.g., hairy cell leukemia, myelomonocytic leukemia). In the state of Massachusetts during the time period 1982–1998, 30.8% of all leukemias diagnosed among females were AML, 23.2% were CLL, 13.8% were ALL, 11.5% were CML, and 20.7% were not classifiable or were other histology types. Among females diagnosed with leukemia in New Bedford CT 6528 during the same time period, three were diagnosed as CLL (37.5%), two were diagnosed as ALL (25.0%), one was diagnosed as CML (12.5%), and two were not classified as one of the four major subtypes (25.0%).

The four main leukemia sub-types have different risk factors suspected to be associated to their development and occur with different frequency among adults and children. ALL occurs predominantly among children (peaking between ages 2 and 3 years). An elevation in the incidence of ALL is also seen among older individuals, which typically begins at approximately 40–50 years of age and peaks at about age 85 (Linet M.S. and Cartwright R.A. 1996). Among females in New Bedford CT 6528, no children were diagnosed with leukemia (of any subtype). Two individuals were diagnosed with ALL. Both were over the age of 65. CLL is chiefly an adult disease; ninety percent of individuals diagnosed with CLL are over the age of 50 (Miller et al. 1990). In New Bedford CT 6528, three females were diagnosed with CLL and all were over the age of 60 with an average age at diagnosis of 80 years. AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS 2000). In addition, it is more common among males than females. In New Bedford CT 6528, no females were diagnosed with AML. CML can occur at any age; however, it is most frequently diagnosed between the ages of 40 and 50 years (ACS 1999). One female was diagnosed with CML in New Bedford CT 6528 during 1982–1998. The remaining two females were not diagnosed with one of the major four sub-types of leukemia but were diagnosed with less common types of leukemia. Therefore, based on the information reviewed, it appears that the majority of leukemia diagnoses among females in this area of New Bedford represented a variety of different cell types with varying sets of risk factors. The average age of diagnosis for all leukemia subtypes combined was 67 years.

(b) Occupation

Several occupational exposures have been identified as playing a role in the development of leukemia. For example, exposures to particular chemicals are thought to increase the risk of developing certain kinds of leukemia. Exposure to ionizing radiation, chronic, high-dose exposure to pesticides, and other chemicals such as benzene, have also been suggested as possible risk factors for leukemia (Linet and Cartwright 1996). Chronic occupational exposure to benzene has been established as a cause of AML. High doses of radiation among survivors of atomic bomb blasts or nuclear reactor accidents are associated with an increased incidence of AML, CML, and ALL, but no association has been established for lower doses such as those used in medical diagnostics.

Among the eight females diagnosed with leukemia in New Bedford CT 6528, occupation was unknown for half of the individuals. Of the remaining four, none indicated a job that has been associated with an increased risk of leukemia.

2. Kidney Cancer

(a) Age and Gender Distribution

While kidney cancer occurred more often than expected in New Bedford CT 6528 overall, in the most recent time period, 1995–1998, males in this area experienced a statistically significant elevation in the incidence of kidney cancer. Females experienced kidney cancer at approximately the rate expected during all three smaller timer periods evaluated.

Kidney cancer most often occurs in the fifth and sixth decades of life (50–70 year age group) and occurs about twice as often in males versus females (ACS 2001). In New Bedford CT 6528, the ages at diagnosis among males during the 1995–1998 time period ranged from 48 to 79 years. The average age at diagnosis (68 years) was consistent with the established age pattern for this cancer type and a higher incidence among older age groups.

(b) Smoking Status

Cigarette smoking is the most important known risk factor for kidney cancer. Smoking increases the risk of developing kidney cancer by 30% to 100% (ACS 2001). In both males and females, a statistically significant dose response relationship between smoking and this cancer type has

been observed. That is, a greater risk of developing kidney cancer exists among individuals who smoke more. Approximately one-third of kidney cancers in men and one-quarter in women may be caused by cigarette smoking (ACS 2001).

Of the five males diagnosed with kidney cancer in CT 6528 during 1995–1998, only one reported a smoking status (non-smoker), and the remaining four had an unknown smoking status. In Massachusetts as a whole, 47% of males diagnosed with kidney cancer during 1995–1998 were current or former smokers at the time of diagnosis, 25% were non-smokers, and 28% had unknown smoking status (see Figure 3). Because smoking status was unknown for four out of the five males diagnosed with this disease in CT 6528 between 1995 and 1998, the role smoking may have played in the development of kidney cancer among these individuals cannot be determined in this report.

(c) Occupation

Although kidney cancer is not generally considered an occupationally associated cancer, some studies have suggested that environmental and occupational factors may be associated with its development. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. In addition, exposure to cadmium is associated with an increased incidence of kidney cancer, particularly among men who smoke. In addition, workplace exposure to organic solvents, such as trichloroethylene (TCE), may increase the risk of this cancer (ACS 2001). More recently, renal cell carcinoma (RCC), the most common type of kidney cancer, has been suggested to be associated with occupational exposure to petroleum, tar, and pitch products. However, studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between exposure to gasoline or other petroleum products and kidney cancer (Lineham et al. 1997; McLaughlin et al. 1996).

Review of occupational information reported to the MCR for the five males diagnosed with kidney cancer in New Bedford CT 6528 during 1995–1998 did not reveal any jobs where exposures associated with the development of kidney cancer would have been likely. One individual reported an occupation as "retired." It is important to note that job title information reported to the MCR is generally limited in nature. However, information gathered from death

records and/or town residence lists indicated that workplace exposures may have been possible for two of these individuals.

C. Analysis of Geographic Distribution of Cancer Incidence

In addition to determining census tract-specific incidence rates for each cancer type, an evaluation was conducted to determine whether any specific cancer type appeared to be concentrated in any one area of New Bedford CT 6528 during the time period evaluated in this report, (1982–1998). Place of residence at the time of diagnosis was mapped for all individuals diagnosed with cancer in this area to assess any possible geographic concentration of cases. To address cancer concerns in relation to Rodney Metals and Brittany Dye, this review also specifically focused on the geographic pattern of cancer cases in neighborhoods close to the two facilities. In addition, the pattern of cancer in the areas surrounding all 21E sites identified in New Bedford CT 6528 was also considered.

As previously mentioned, cancer is one word that describes many different diseases. Therefore, the occurrence of different types of cancer among individuals in a particular geographic area is not indicative of an unusual or atypical pattern. For the purposes of this evaluation, the geographic distribution of each cancer type was evaluated separately to determine whether an atypical pattern of any one type occurred. In addition, cancers that may be associated with specific environmental exposures of concern were also evaluated geographically to determine whether any atypical patterns of cases exist that may suggest an association with a potential environmental factor.

For the majority of cancer types evaluated, review of the geographic distribution of cancer diagnoses in New Bedford CT 6528 revealed no apparent spatial patterns at the neighborhood level that could not be attributed to factors such as areas of higher population density. For example, although a statistically significant elevation of leukemia among females was observed in this area during 1982–1998, the diagnoses were fairly evenly distributed throughout CT 6528 and seemed to coincide closely with the pattern of population in these areas. However, the five males diagnosed with kidney cancer during 1995–1998 appeared to be concentrated in the northeast corner of the census tract where both Rodney Metals and Brittany Dye are located. Specifically, all five males diagnosed with kidney cancer between the years 1995–1998 resided

in relatively close proximity to these facilities at the time of diagnosis. Review of specific case information available through the MCR did not suggest a single common risk factor for these individuals other than place of residence. Finally, no apparent geographic concentrations of individuals with cancer were noted in areas near any of the other MDEP 21E sites.

VII. DISCUSSION

Six of the eight cancer types evaluated in this report in New Bedford CT 6528 during the time period 1982–1998 were approximately at or slightly above or below the expected rates. Statistically significant elevations in incidence were observed for leukemia among females in this area during the overall time period 1982–1998. In addition, males experienced kidney cancer more often than expected in the most recent time period 1995–1998. This elevation was also statistically significant. However, a number of cancer types occurred less often than expected in New Bedford CT 6528. For example, lung cancer occurred statistically significantly less often than expected based on state rates during 1982–1998.

In addition to the cancer incidence analyses performed in New Bedford CT 6528, an analysis of available information to determine possible sources of environmental contamination in this area and whether these sources could result in environmental exposures to nearby residents was conducted. As a result of this part of the investigation, it was determined that some of the chemicals used at Brittany Dye and, at one time, Rodney Metals belong to a class of compounds called chlorinated volatile organic compounds (VOCs). These compounds are found throughout the environment mostly as a result of industrial activities (ATSDR 1997). Due to some of their chemical characteristics (high vapor pressures), VOCs easily disperse into air and, therefore, the most likely way for people to be exposed to these chemicals is volatilization to air. Exposures to VOCs are typically expected to occur in certain individuals and under specific conditions. Some examples include inhalation exposures to workers involved in degreasing operations, inhalation and ingestion exposures to individuals who use private wells located near disposal/contamination sites, and inhalation by consumers who use products containing VOCs in areas of poor ventilation (Wu and Schaum 2000).

At Rodney Metals, two specific VOCs, TCE and 1,1,1-TCA, as well as several of their chlorinated breakdown products (i.e., 1,1-DCE and vinyl chloride) were detected in groundwater below the surface as well as in surface water samples taken from New Bedford Harbor. Sampling of groundwater monitoring wells, MW-106 and MW-107, situated on the eastern-northeastern side of the Rodney Metals facility, away from residential neighborhoods, showed the highest concentrations of TCE and 1,1,1-TCA. These two chlorinated VOCs were detected at levels exceeding health-based guidelines for drinking water. However, although TCE, 1,1,1-TCA and several of their breakdown products exceeded drinking water screening values in groundwater in this area, exposure to contaminated groundwater is unlikely for individuals residing in neighborhoods abutting the Rodney Metals facility. As discussed, groundwater in this area is not being used for drinking water purposes, and therefore, ingestion of contaminated groundwater would not be expected. In addition, groundwater underneath the Rodney Metals facility flows in an east-northeasterly direction away from residences adjacent to the facility. Therefore, exposure to VOCs through inhalation and dermal contact related to groundwater also would not be expected to occur for residents who live near the facility. Further, even though chemical contaminants may be present at concentrations exceeding health based comparison values or standards, they are located below ground surface, and contact with either the chemicals themselves or the environmental media where these chemicals were detected is not likely. Based on this information, it is unlikely that adverse health effects associated with subsurface contamination identified at the Rodney Metals facility would occur for nearby residents.

Like VOCs, there are a variety of ways that polycyclic aromatic hydrocarbons (PAHs) enter the environment. For example, PAHs are contained in tobacco smoke, wood smoke, and a variety of foods, particularly smoked foods. As a result, the greatest sources of exposure to PAHs for most of the United States population are through contaminated air and ingestion of the compounds in foodstuffs, including cereals, grains, flour, bread, vegetables, fruits, meat, and processed or pickled foods. In addition, the general population may be exposed to PAHs in drinking water, through skin contact with soot and tars, and via ingestion and inhalation (during cooking) of grilled or smoked foods (ATSDR 1995a). Mineral oil and several of its constituents, including toluene and a variety of PAHs that included benzo(a)pyrene, fluoranthene, and naphthalene were detected in groundwater and subsurface soil at the Rodney Metals facility. Although some of these compounds were detected at concentrations exceeding health-based/clean-up standards, the

area of contamination was confined to the northeast corner of the facility. Similar to the VOCs detected on the eastern-northeastern side of the Rodney Metals facility, direct contact with the mineral oil (and the PAHs and toluene present in the mineral oil) by individuals residing in neighborhoods abutting the facility appears to be unlikely. This is because the contamination is located below the surface, and groundwater flows away from the residential areas.

Therefore, although releases of contaminants to the subsurface occurred at Rodney Metals in the past, currently, there is no information to suggest that the contaminants detected in subsurface media at the Rodney Metals facility and surface water across East Rodney French Boulevard are posing a health threat to individuals visiting or residing in neighborhoods adjacent to the facility.

It has been reported that stack and/or fugitive releases of VOCs to the air from both Rodney Metals and Brittany Dye have occurred in the past. Specifically, review of TRI release data from 1987 to 2000 indicates that Rodney Metals released 1,1,1-TCA to the air prior to 1996. Brittany Dye has reported release of 1,1,1-TCA to the air prior to 1995 and release of TCE to the air for the years 1995 to 2000 (USEPA 2003). However, there are no sampling data available to determine whether these emissions resulted in concentrations of VOCs in the ambient air in the vicinity of these facilities. While available information indicates that the prevailing wind direction in the city of New Bedford is generally from the west (City of New Bedford 2003), specific data on environmental conditions such as wind direction and speed in areas immediately surrounding Rodney Metals and Brittany Dye were unavailable. Therefore, causal conclusions cannot be drawn regarding whether or not emission releases from the two facilities, particularly emissions of chlorinated VOCs, could contribute to the development of health effects reported by individuals living in the vicinity of the two facilities of concern. Nevertheless, although it is not possible to determine whether individuals residing in the vicinity of Rodney Metals and Brittany Dye are being exposed to air emissions, information pertaining to possible health effects from inhalation exposure to TCE and 1,1,1-TCA was further investigated.

Occupational studies of workers exposed to unmeasured levels of TCE in air have been unable to provide definitive evidence for an increased cancer risk and are often limited by multiple chemical exposures and small numbers of study participants. In general, evidence that TCE is carcinogenic to humans is inconclusive and requires further study. While some studies have

shown no association between inhalation exposure to TCE and cancer, others have found slight increases in a number of cancer types including cancers of the kidney, stomach, liver, prostate, bladder and NHL. However, problems with study design were often reported, and associations were often based on small numbers of individuals and complicated by confounding factors (ATSDR 1997).

As observed in several toxicological investigations in experimental mice and rats, TCE has been shown to cause liver, lung, and kidney cancer and, to a lesser extent, lymphomas and leukemia; however, there are limitations to using these types of studies when evaluating potential health effects to humans. For example, animal studies frequently use relatively high concentrations of chemicals that are several orders of magnitude higher than those to which humans may be exposed either in occupational or environmental settings and therefore, often do not represent human exposure conditions (Wartenberg et al. 2000, Lash et al. 2000, ATSDR 1997).

Available information does not indicate that 1,1,1-TCA causes cancer. No studies were located that showed an association between inhalation of 1,1,1-TCA among humans and cancer. Studies conducted in both rats and mice exposed to 1,1,1-TCA in air demonstrated no evidence of cancer health effects (ATSDR 1995b).

As discussed, although kidney cancer in New Bedford CT 6528 occurred approximately at or below expected rates during the two earlier time periods examined (1982–1986 and 1987–1994), the incidence of kidney cancer among males was statistically significantly elevated in CT 6528 during the most recent time period evaluated 1995–1998. Furthermore, the individuals that were diagnosed with this disease during this time period resided, at the time of diagnosis, within relatively close proximity to the two facilities evaluated in this report. Individuals diagnosed with kidney cancer during the earlier time periods were generally more evenly distributed throughout the census tract. As discussed, although somewhat inconclusive, some epidemiologic evidence has shown support for an association between occupational exposure to TCE and kidney cancer, liver cancer, and, to a lesser extent, for NHL. Therefore, if the incidence of kidney cancer in this area of the South End of New Bedford were to be attributed to an environmental exposure to TCE, then it might be expected that NHL and, in particular, liver cancer also would be occurring at a greater incidence in this area. Close review of the spatial

distribution of these diseases in relation to Rodney Metals and Brittany Dye revealed no unusual patterns of liver cancer or NHL in the vicinity of the two facilities throughout the entire 17-year period evaluated; in general, diagnoses of liver cancer and NHL were fairly evenly distributed throughout CT 6528. Also, the incidence of liver cancer and NHL in CT 6528 was either less than or equal to the expected rates during 1982–1998.

Leukemia among females also occurred at a rate that was statistically significantly elevated in CT 6528 from 1982–1998. However, among males, the incidence of leukemia was approximately equal to the rate expected during this time. The pattern of leukemia did not indicate a concentration or an atypical distribution of individuals diagnosed in the area surrounding Rodney Metals or Brittany Dye, and most individuals did not reside in this area of the census tract. No clear evidence, in experimental animal or human studies, points to an association between exposure to TCE in ambient air and the development of leukemia. While there is some evidence in the epidemiologic literature that suggests an association between exposure to TCE in drinking water (when mixed with other contaminants) and childhood leukemia (ATSDR 1997, MDPH 1997, Cohn et al. 1994, Fagliano et al. 1990, Wartenberg et al. 2000), the groundwater in the area of Rodney Metals and Brittany Dye is not a drinking water source. Further, there were no cases of childhood leukemia in New Bedford CT 6528 where Rodney Metals and Brittany Dye are located. For these reasons, it does not appear likely that the increased occurrence of leukemia observed among females residing in New Bedford CT 6528 from 1982–1998 is likely associated with past or current activities related to the two facilities. Moreover, as previously discussed, leukemia is not one type of cancer but is classified into four main types of disease; each type of leukemia has different risk factors suspected to be associated with their development. In CT 6528, a variety of different types of leukemia were diagnosed among individuals in this area with no pattern of any one type occurring. Therefore, this information suggests that leukemia among females in CT 6528 is not likely related to a common environmental factor in this area but more likely related to the presence of individual known or suspected risk factors for the specific subtypes of leukemia among these persons.

For the majority of cancer types evaluated, analysis of the geographic distribution of residences of individuals in New Bedford CT 6528 diagnosed with cancer during 1982–1998 did not reveal any atypical spatial patterns of disease. Specifically, the distribution of cancer diagnoses varied

geographically and seemed to coincide closely with the pattern of population in this census tract. However, males diagnosed with kidney cancer during the most recent time period evaluated appeared to be concentrated in the northeast corner of the census tract. Specifically, all five individuals diagnosed with kidney cancer between 1995–1998 were located in close proximity to the two facilities. No apparent geographic concentrations of individuals with cancer were noted in areas near any of the other MDEP 21E sites.

Available risk factor information for cancer types that demonstrated a statistically significant elevation during 1982–1998, leukemia and kidney cancer, was compared to known or established incidence trends to assess whether any unexpected patterns exist in New Bedford CT 6528. The age distribution of individuals diagnosed with these cancer types was generally consistent with disease patterns described in the epidemiological literature. Review of available information from the MCR on smoking indicated that smoking status was unknown for four out of five of the individuals diagnosed with kidney cancer during the most recent time period. While information reported to the MCR on occupation for these five individuals was somewhat limited, information gathered from death records and/or town residence lists indicated that workplace exposures may have contributed to the disease for two of these individuals. Of these five males diagnosed with kidney cancer during this time period, three had lived at their address for more than 30 years at the time of their cancer diagnosis, one was a short-term resident of the area, and residential history information could not be confirmed for the remaining individual, making it more likely that he lived at the reported address for a short period of time. Finally, the role that other personal risk factors, such as genetics and diet, may have played in the incidence of kidney cancer among these individuals could not be evaluated in this report.

Residents of the South End area of New Bedford near Rodney Metals and Brittany Dye also conveyed concerns about acute non-cancer health outcomes, such as upper respiratory irritation, nausea, and headaches. In 1995, Rodney Metals switched to cleaner technologies to reduce waste generation, and degreasing operations using chlorinated VOCs were replaced by aqueous based processes using several acidic chemicals like hydrofluoric and nitric acids (USEPA 2003). TRI data indicates that from 1995 to 2000 Brittany Dye emitted TCE and prior to 1995, the facility emitted 1,1,1 TCA. As a result, individuals in the area immediately surrounding the facilities have the potential to detect several distinctive odors attributed to these chemicals. For

example, hydrofluoric and nitric acids have strong acidic, irritating odors; while several of the chlorinated VOCs, namely TCE and 1,1,1-TCA, have overpoweringly sweet, chloroform-like odors (HSDB 2002a-e). In addition, it is possible that some residents living in close proximity to the two facilities could experience some irritant effects associated with VOCs in ambient air. It is important to note that some individuals, particularly those with pre-existing conditions such as asthma and allergies may experience irritant reactions that would not necessarily impact the general population similarly. Studies of workers chronically exposed to TCE in air have reported sleepiness, dizziness, headaches, and nausea, and there is some suggestion that people who breath high levels of TCE may develop damage to the nerves in the face (ATSDR 1997). Inhalation of air containing high levels of 1,1,1-TCA for a short period of time can cause dizziness, lightheadedness and a possible loss of coordination (ATSDR 1995b).

Based on the environmental information reviewed in this evaluation, including environmental data for the Rodney Metals and Brittany Dye facilities, it does not appear that environmental exposures played a major role in the incidence of most cancers in New Bedford CT 6528 during the 17-year time period 1982–1998. However, it is important to note that data regarding ambient air concentrations of emissions associated with these two facilities are not available. Therefore, without these data, it is not possible to evaluate whether there are elevated levels of VOCs present in the ambient air adjacent to these facilities and whether they may be contributing to cancer or non-cancer health effects experienced by residents in this area. In addition, this evaluation cannot determine the exact cause (either environmental or otherwise) of any one individual's cancer diagnosis. Although some individuals diagnosed with kidney cancer were more geographically concentrated in the vicinity of the Rodney Metals and Brittany Dye facilities, it is not possible to determine whether exposures from these two facilities may in fact be related to the development of kidney cancer among these individuals.

VIII. CHILD HEALTH ISSUES

ATSDR and MDPH, through ATSDR's Child Health Initiative, recognize that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their environment. Children are at a greater risk than adults from certain kinds of exposure to hazardous substances emitted from waste sites. They are more likely to be

exposed because they play outdoors and because they often bring food into contaminated areas. Because of their smaller stature, they might breathe dust, soil, and heavy vapors close to the ground. Children are also smaller, resulting in higher doses of contaminant exposure per body weight. The developing body systems of children can sustain permanent damage if certain toxic exposures occur during critical growth stages. Most importantly, children depend completely on adults for risk identification and management decisions, housing decisions, and access to medical care. The incidence and patterns of cancer among children in New Bedford CT 6528 is discussed in Section VI ("Analysis of Cancer Incidence in New Bedford CT 6528") of this report. No exposures were identified that would indicate that children are being impacted by the facilities; however, air data are not available to determine whether exposures to chemicals released into the air could be possible.

IX. LIMITATIONS

This assessment is an investigation that analyzes descriptive health outcome data for cancer to determine whether the pattern or occurrence of selected cancers is unusual. The purpose of this investigation is to evaluate the patterns of cancer in a geographical context in relation to available information about factors, including environmental factors, related to cancer to see whether further investigation seems warranted. Information from descriptive analyses, which may suggest that a common etiology (or cause) is possible, can serve to identify areas where further public health actions may be warranted. Inherent limitations in this type of analysis and the available data make it impossible to determine the precise causal relationships or synergistic roles that may have played a part in the development of individual cancers in these communities. Also, this type of analysis cannot determine what may have caused any one individual's cancer. Cancers in general have a variety of risk factors known or suggested to be related to the etiology (cause) of the disease that could not be evaluated in this report. It is believed that many cancers are related largely to behavioral factors such as cigarette smoking, diet, and alcohol consumption. Other factors associated with cancer are socioeconomic status, heredity/genetics, race, and geography. It is beyond the scope of this report to determine the causal relationship of these factors and the development of cancer or other health outcomes in New Bedford CT 6528.

Like the analysis of descriptive health outcome data, there are several limitations encountered when analyzing the environmental data. As a result, these limitations make it impossible to determine the role potential exposures to specific contaminants or to environmental media harboring those contaminants may have played in the development of an individual's cancer. That is, due to historical and analytical data gaps in the environmental data, this type of evaluation cannot conclude what may have caused any one individual's cancer, whether it be environmental, behavioral, viral, genetic or a combination of these elements.

X. CONCLUSIONS

- ATSDR requires that one of five conclusion categories be used to summarize findings of a health consultation. These categories are as follows: (1) Urgent Public Health Hazard; (2) Public Health Hazard; (3) Indeterminate Public Health Hazard; (4) No Apparent Public Health Hazard; (5) No Public Health Hazard. A category is selected from site-specific conditions such as the degree of public health hazard based on the presence and duration of human exposure, contaminant concentration, the nature of toxic effects associated with site-related contaminants, presence of physical hazards, and community health concerns. Although no indication was found that people are currently exposed to chemicals from the Rodney Metals or Brittany Dye facilities, data regarding ambient air concentrations of emissions associated with these two facilities are unavailable making it difficult to evaluate whether chemicals are being released into the air that might be harmful. Therefore, ATSDR classifies the Rodney Metals and Brittany Dye facilities, in the past, present, and future, as posing an Indeterminate Public Health Hazard.

- Of the eight cancer types evaluated in the community of New Bedford CT 6528 during 1982–1998, the majority occurred approximately at or near the expected rate. The exceptions included statistically significant elevations in the incidence of leukemia among females during the overall time period 1982–1998 and kidney cancer among males during the most recent time period 1995–1998. The rate of lung cancer in this area was statistically significantly lower than expected during this time.

- With the exception of kidney cancer, review of the geographic distribution of cancer in New Bedford CT 6528 revealed no apparent spatial patterns at the neighborhood level that could not be attributed to factors such as areas of higher population density. Further, no unexpected concentrations of diagnoses were observed close to the Rodney Metals and Brittany Dye properties or other environmental release sites in this area.

- A concentration of five individuals diagnosed with kidney cancer was observed within close proximity to the Rodney Metals and Brittany Dye facilities. Available data on smoking and occupation were limited, and the possible role of these and other risk factors could not be evaluated for these individuals. It is important to note, however, that contaminants of concern have been suggested in some studies to be associated with kidney cancer, liver cancer, and, to a lesser extent, NHL. Neither NHL nor liver cancer displayed similar geographic patterns of incidence.

- In the past, two releases of mineral oil and one release of chlorinated VOCs occurred at the Rodney Metals facility. However, based on the data reviewed, it does not appear that individuals living in the vicinity of Rodney Metals are being exposed to this subsurface contamination, and these releases are not likely related to cancer among residents of the surrounding neighborhoods. Chlorinated VOCs were also detected in surface water in New Bedford Harbor. However, based on the levels of VOCs detected in surface water, the potential for adverse health effects associated with recreational use of surface water is not likely.

- No ambient air sampling data exist, and therefore, it cannot be determined if individuals residing in the vicinity of Rodney Metals and Brittany Dye are being exposed to elevated levels of chlorinated VOCs, namely TCE and 1,1,1-TCA. However, for seven of the eight cancer types evaluated, no unusual patterns of incidence or unexpected trends were observed in this area of New Bedford.

- Residents of the South End area of New Bedford near Rodney Metals and Brittany Dye also conveyed concerns about acute non-cancer health outcomes, such as upper respiratory irritation, nausea, and headaches. It is possible that some residents living in close proximity to these facilities could experience some irritant effects associated with

VOCs in ambient air or nuisance odors, particularly those with pre-existing medical conditions such as asthma or allergies. Such individuals may experience irritant reactions that would not necessarily impact the general population similarly.

- Analysis of environmental and non-environmental risk factors, as well as an evaluation of the geographic distribution of cases, did not reveal a clear pattern suggesting that environmental exposures to chemicals from Rodney Metals or Brittany Dye contributed to the incidence of most cancers in New Bedford CT 6528 during the 17-year time period 1982–1998. Moreover, the available data do not show a common pattern that would suggest that any single risk factor (environmental or otherwise) is likely to be responsible for the incidence of cancer in this community. Rather, a combination of factors such as smoking, diet, and exercise may be contributing to incidence rates in this area of New Bedford.

XI. RECOMMENDATIONS

- MDPH should further characterize opportunities for exposure if additional environmental data on air emissions or ambient air quality in the vicinity of Rodney Metals and Brittany Dye become available.

- Due to the unpleasant odors and nuisance conditions being reported by individuals residing in and visiting areas surrounding the two facilities, MDPH recommends that the MDEP work with both Rodney Metals and Brittany Dye to determine any additional actions that could reduce potential impacts to residents in the surrounding neighborhoods.

- The New Bedford Health Department should continue to work with the community and the BEHA's Environmental Health Education Program to provide educational information and conduct outreach activities to New Bedford residents about ways to reduce their risk of cancer. This could be in concert with ongoing health education and outreach activities currently underway by MDPH and EPA in New Bedford.

- The MDPH/BEHA should continue to monitor the incidence of all cancer types in the city of New Bedford through the Massachusetts Cancer Registry.

XII. PUBLIC HEALTH ACTION PLAN

The Public Action Plan for the neighborhood surrounding Rodney Metals and Brittany Dye in the South End of New Bedford, Massachusetts, contains recommendations for actions to be taken at and in the vicinity of the above-mentioned facilities subsequent to the completion of this health consultation on cancer incidence assessment and possible exposure to chemicals released from Rodney Metals and Brittany Dye. The purpose of the Public Health Action Plan is to ensure that this health consultation not only identifies potential public health hazards, but also provides a plan of action designed to mitigate and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included is a commitment on the part of the ATSDR/MDPH to follow up on this plan to ensure that it is implemented. The public health actions to be implemented by ATSDR/MDPH are as follows:

- If additional environmental data on air emissions or ambient air quality in the vicinity of Rodney Metals and Brittany Dye become available, MDPH will continue efforts to provide a more comprehensive review of potential exposures to residents living near these facilities.

- Based on the observed geographic concentration of kidney cancer diagnoses in proximity to the Rodney Metals and Brittany Dye facilities during the most recent time period, 1995–1998, the MDPH will provide additional follow-up for all individuals diagnosed with this cancer type in New Bedford CT 6528 over the seventeen years evaluated, 1982–1998. Specifically, these 12 individuals (or their families) who provide informed consent will have the opportunity for personal interviews and/or medical records review by an environmental/occupational physician to determine any possible environmental or other factors that may have contributed to their diagnosis.

- The MDPH/BEHA will continue to monitor the incidence of all cancer types in the city of New Bedford through the Massachusetts Cancer Registry.

- At the request of the New Bedford Health Department, BEHA's Environmental Health Education Program could work to incorporate activities aimed at cancer risk reduction into their existing education and outreach efforts.

- ATSDR and MDPH will reevaluate and expand the Public Health Action Plan when needed. New environmental, toxicological, or health outcome data may determine the need for additional actions at the focus area.

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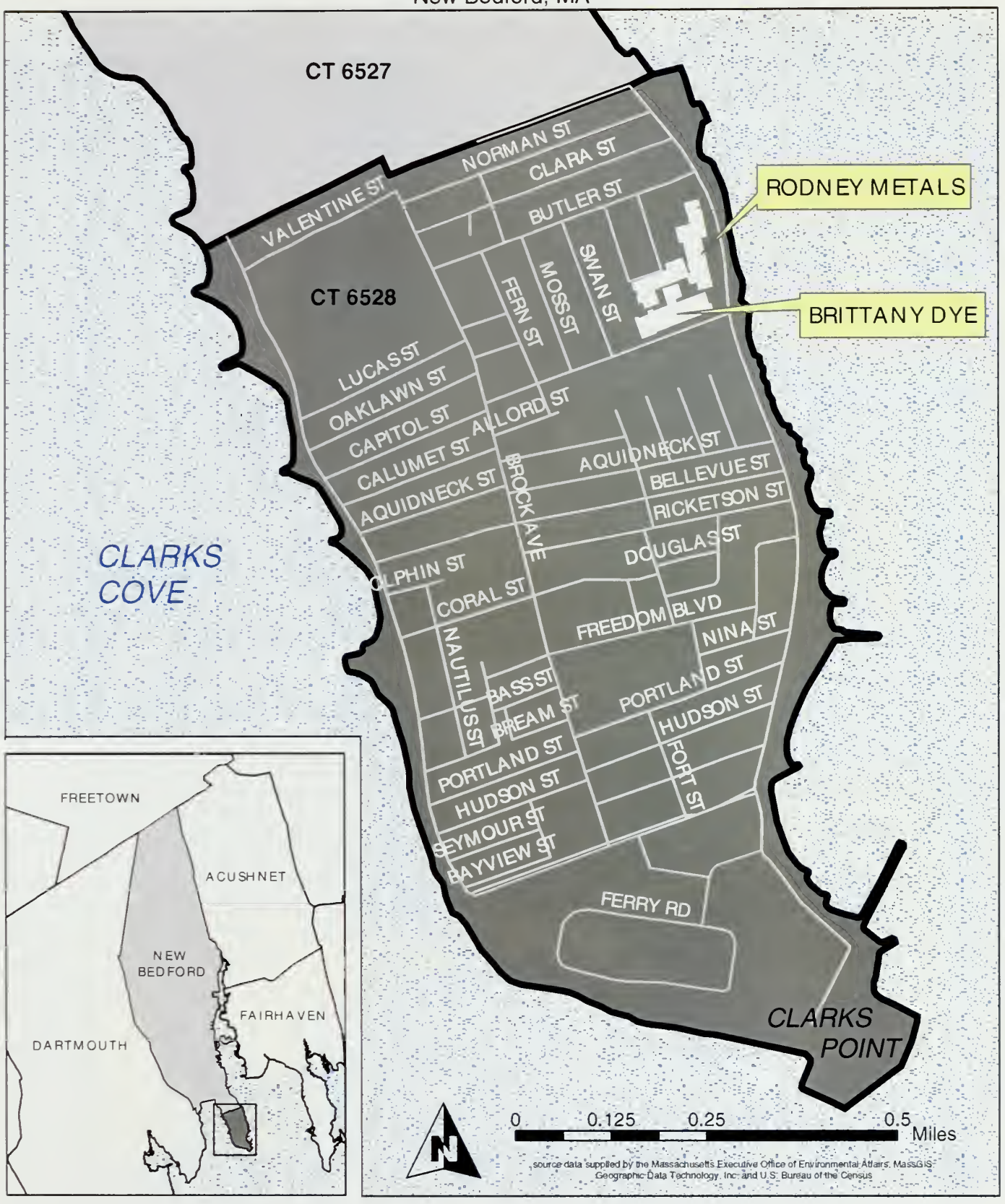
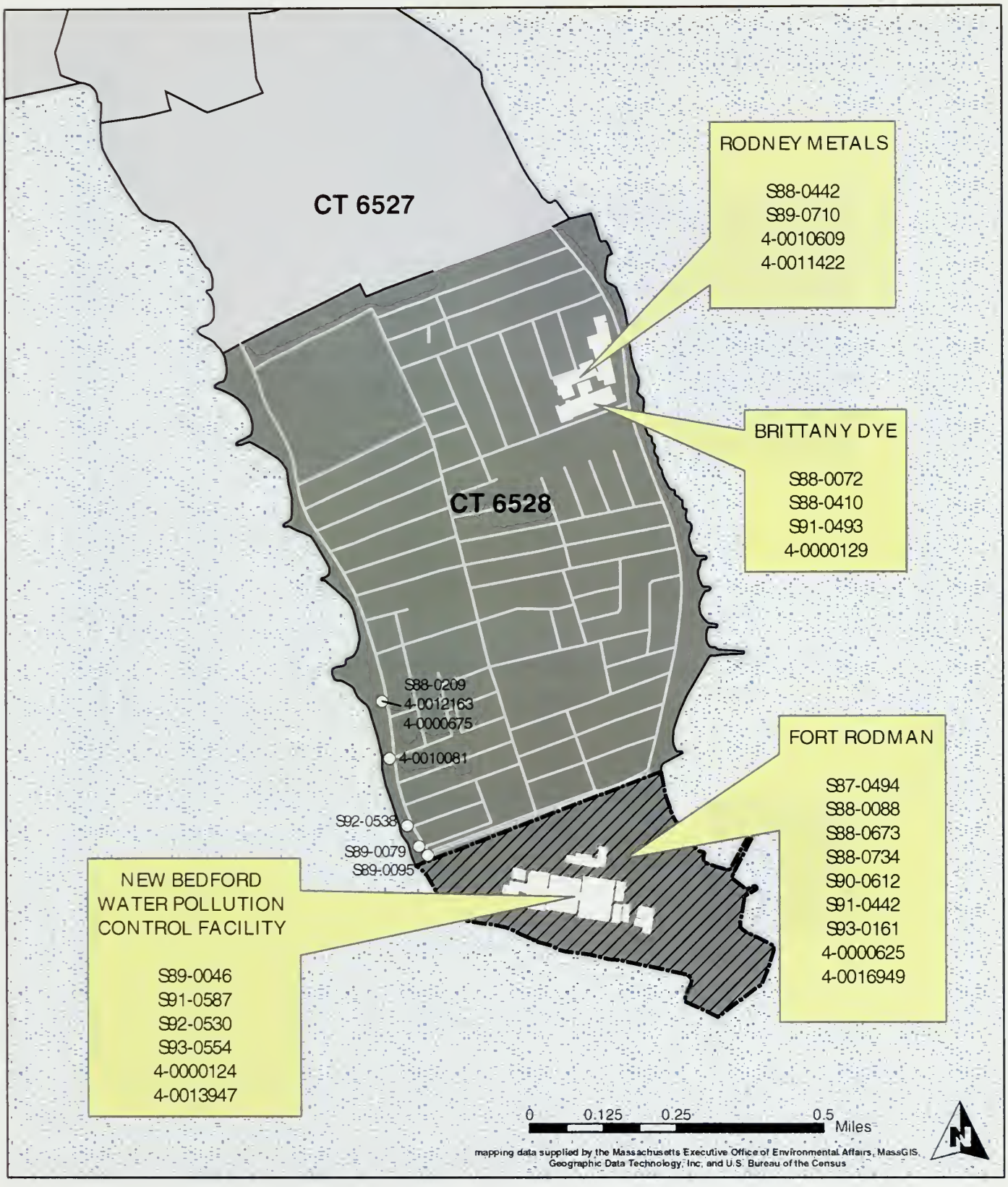
FIGURES

Figure 1
Census Tract (CT) 6528
New Bedford, MA



Bureau of Environmental
BEHA
Health Assessment

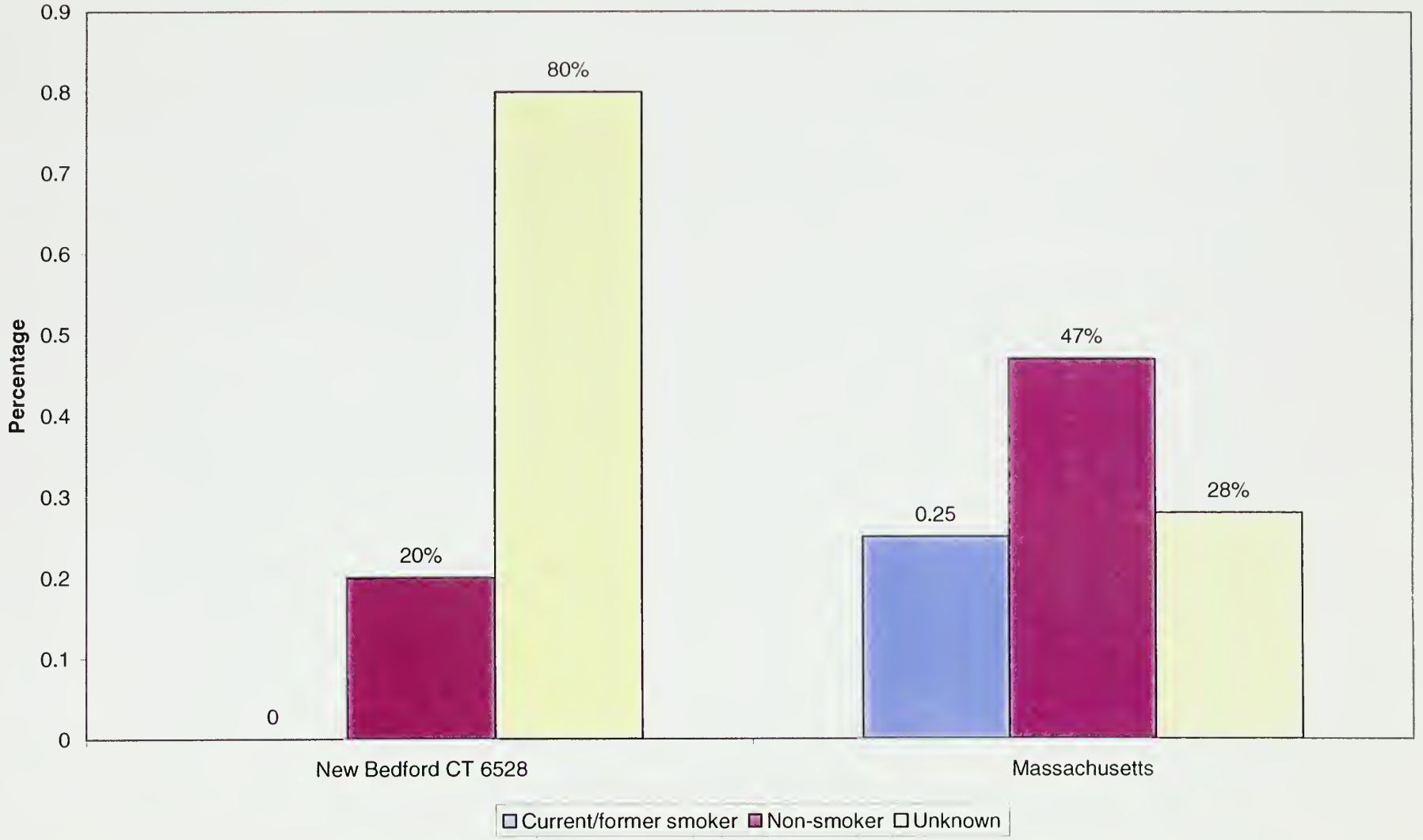
Figure 2
Approximate Locations of MDEP 21E Hazardous Waste Sites
Census Tract (CT) 6528
New Bedford, MA



Bureau of Environmental Health Assessment

Approximate Location of 21E Site
Street
Census Tract (CT) 6528
Fort Rodman
Ocean

Figure 3: Smoking Status of Males with Kidney Cancer in New Bedford CT 6528 and Massachusetts, 1995-1998



TABLES

Table 1
MDEP 21E Hazardous Material and Oil Releases
Census Tract 6528
New Bedford, MA

SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES
	4-0000129	TELEDYNE BRITTANY DYE	E RODNEY FRENCH BLVD	8/15/1985	UNKNOWN CHEMICAL OF UNKNOWN TYPE	
	4-0000124	NEW BEDFORD WWTP	CLARKS POINT	1/15/1987	UNKNOWN CHEMICAL OF UNKNOWN TYPE	
S87-0494			FORT RODMAN	8/8/1987	WASTE OIL (10-15 GALLONS)	DRUM
S88-0072		BRITTANY DYE	RODNEY FRENCH BLVD	2/12/1988	UNKNOWN	
S88-0410			BRITTANY DYE	6/30/1988	DYE	DRUM
S88-0673		FORT RODMAN		10/22/1988	SEWAGE	
	4-0000625	US ARMY RESERVE CTR	FORT RODMAN	1/15/1989	UNKNOWN CHEMICAL OF TYPE - OIL	UST
S89-0046		N.B. WASTE WATER TREATMENT PLANT		1/27/1989	WASTE OIL (101-250 GALLONS)	
S89-0079		3 TENANT HOUSE	13 RODNEY FRENCH BLVD	2/6/1989	#2 FUEL OIL (1-10 GALLONS)	PIPE/HOSE/LINE
S89-0095			#3 RODNEY FRERENCH BLVD	2/8/1989	#2 FUEL OIL	PIPE/HOSE/LINE
	4-0000675	FURNITURE CITY	127 W RODNEY FRENCH BLVD	4/15/1989	UNKNOWN CHEMICAL OF UNKNOWN TYPE	
S89-0710		TELEDYNE RODNEY METAL	1357 E RODNEY FRENCH BLVD	10/13/1989	TRICHLOROETHANE (111)	OTHER SOURCE (DEGREASER)
S90-0612	4-0000625	US ARMY RESERVE CTR	FORT RODMAN	8/16/1990	#2 FUEL OIL	U.S.T.
S91-0442		FORT RODMAN	RODNEY FRENCH BLVD	7/26/1991	DIESEL FUEL	ABOVE-GRND TANK
S91-0493		BRITTANY DYE	E RODNEY FRENCH BLVD & BUTLER	8/20/1991	OTHER MATERIAL (WASTEWATER/DYE)	PIPE/HOSE/LINE
S91-0587		N.B. WATER POLL CNTRL FAC	FORT RODMAN	9/24/1991	GASOLINE	----------
S92-0538		PROPERTY	31 RODNEY FRENCH BLVD	7/16/1992	OTHER MATERIAL (UNKNOWN)	U.S.T.
S92-0530		NB WASTEWATER TREATMENT PLANT	FORT RODMAN	7/29/1992	OTHER MATERIAL (SEWAGE)	OTHER SOURCE (WWTP)
S93-0161		WASTEWATER TREATMENT PLANT	FORT RODMAN	3/10/1993	CHLORINE	HOSE
S93-0554		WWTP MA MILITARY RESERVATION	FORT RODMAN	8/11/1993	OTHER MATERIAL (SEWAGE)	TANKER TRUCK
	4-0010081	REAR OF PROPERTY	89 WEST RODNEY FRENCH BLVD	11/9/1993	DIESEL FUEL (100 GAL)	PIPE
	4-0010609	TEDEDYNE RODNEY METALS	1357 EAST RODNEY FRENCH BLVD	7/15/1994	UNKNOWN CHEMICAL OF TYPE - OIL (32000 MG/KG); TOTAL PETROLEUM HYDROCARBONS (TPH) (32000 PPM); TOTAL PETROLEUM HYDROCARBONS (TPH) (3000 PPM)	UNKNOWN
	4-0011422	TELEDYNE RODNEY METALS	1357 RODNEY FRENCH BLVD	5/31/1995	LUBRICATING OIL (36 INCH); MINERAL OIL (3.9)	UNKNOWN
	4-0012163	NO LOCATION AID	127 WEST RODNEY FRENCH BLVD	5/6/1996	DIESEL FUEL (15 GAL)	SADDLETNK
	4-0013947	WWTP DEMOLITION	1000 WEST RODNEY FRENCH BLVD	6/15/1998	HYPOCHLOROUS ACID, SODIUM SALT (400 GAL)	AST

SPILL ID	RTN	LOCATION AID	ADDRESS	DATE	MATERIALS	SOURCES
	4-0016949	FMR NAVAL RESERVE CENTER	838 SOUTH RODNEY FRENCH BLVD	3/15/2002	FUEL OIL #2 (0.05 GAL)	
S88-0088		BUILDING 17	US A.R.C. FORT RODMAN		#2 FUEL OIL	U.S.T.
S88-0209		FURNITURE CITY	127 WEST RODNEY BLVD		#6 FUEL OIL	U.S.T.
S88-0442			1357 EAST RODNEY FRENCH BLVD		UNKNOWN	
S88-0734	4-0000625	ARMY RESERVE CENTER	HHC, 483 ENG FORT RODMAN		#2 FUEL OIL	U.S.T.

Data Source: MDEP Bureau of Waste Site Cleanup. 2003. Downloadable Site Lists. http://www.state.ma.us/dep/bwsc/sites/sdown.htm

Notes:
Spill ID - Spill Identification Number (applicable for spills reported prior to October 1993)
RTN - Release Tracking Number. Unique ID number assigned to spills not remediated by October 1993 and to those occuring October 1993-present
Location Aid - Place name of spill
Address - Street location of spill
Date - Date of spill (spills prior to October 1993), or date spill was reported to MDEP (for spills occurring October 1993-present)
Materials - Chemical(s) found at site
Sources - Origin(s) of contamination at site

TABLE 2
Summary of Groundwater Sample Analytical Results
Rodney Metals, New Bedford, Massachusetts

COMPOUND	SAMPLING LOCATION	MAXIMUM DETECTED CONCENTRATION	DRINKING WATER COMPARISON VALUE	EPA MAXIMUM CONTAMINANT LEVEL (MCL)
Volatile Organic Compounds (VOCs)				
Chlorobenzene	MW-103	2 ug/L	Intermediate EMEG (adult) = 10,000 ppb Intermediate EMEG (child) = 4,000 ppb RMEG (adult) = 700 ppb RMEG (child) = 200 ppb	100 ppb
1,1-Dichloroethane	MW-103	17 ug/L	MDEP GW-2 Standard = 9,000 ug/L	NA
1,1-Dichloroethene	MW-107	30,000 ug/L	Chronic EMEG (adult) = 300 ppb Chronic EMEG (child) = 90 ppb	7 ppb
cis-1,2-Dichloroethene	MW-108	66 ug/L	Intermediate EMEG (adult) = 10,000 ppb Intermediate EMEG (child) = 3,000 ppb	70 ppb
trans-1,2-Dichloroethene	MW-201	1 ug/L	Intermediate EMEG (adult) = 7,000 ppb Intermediate EMEG (child) = 2,000 ppb RMEG (adult) = 700 ppb RMEG (child) = 200 ppb	100 ppb
Methylene chloride	MW-106	3,200 ug/L	CREG = 5 ppb Chronic EMEG (adult) = 2,000 ppb Chronic EMEG (child) = 600 ppb	5 ppb
Tetrachloroethene	MW-101	3,200 ug/L	RMEG (adult) = 400 ppb RMEG (child) = 100 ppb	5 ppb
Toluene	MW-101	100,000 ug/L	Intermediate EMEG (adult) = 700 ppb Intermediate EMEG (child) = 200 ppb RMEG (adult) = 7,000 ppb RMEG (child) = 2,000 ppb	1,000 ppb
1,1,1-Trichloroethane	MW-107	200,000 ug/L	LTHA = 200 ppb	200 ppb
Trichloroethene	MW-107	29,000 ug/L	NA	5 ppb
Vinyl chloride	MW-201	7 ug/L	CREG = 0.03 ppb Chronic EMEG (adult) = 0.7 ppb Chronic EMEG (child) = 0.2 ppb	2 ppb
Polycyclic Aromatic Hydrocarbons (PAHs)				
Benzo(a)anthracene	MW-101	1,800 ug/L	NA	NA
Benzo(a)pyrene	MW-101	730 ug/L	CREG = .005 ppb	0.2 ppb
Chrysene	MW-101	3,500 ug/L	NA	NA
Fluoranthene	MW-101	130,000 ug/L	Intermediate EMEG (adult) = 10000 Intermediate EMEG (child) = 4000	NA
Fluorene	MW-101	12,000 ug/L	Intermediate EMEG (adult) = 10000 Intermediate EMEG (child) = 4000	NA
2-Methylnaphthalene	MW-101	9,500 ug/L	MDEP GW-3 Standard = 3,000 ug/L	NA

TABLE 2
Summary of Groundwater Sample Analytical Results
Rodney Metals, New Bedford, Massachusetts

COMPOUND	SAMPLING LOCATION	MAXIMUM DETECTED CONCENTRATION	DRINKING WATER COMPARISON VALUE	EPA MAXIMUM CONTAMINANT LEVEL (MCL)
Naphthalene	MW-101	2,900 ug/L	Intermediate EMEG (adult) = 700 ppb Intermediate EMEG (child) = 200 ppb RMEG (adult) = 700 ppb RMEG (child) = 200 ppb	140 ug/L *
Phenanthrene	MW-101	82,000 ug/L	NA	NA
Pyrene	MW-101	12,000 ug/L	NA	NA

Data Source: Innovative Engineering Solutions, Inc. 2002. Phase II: Comprehensive Site Assessment and Phase II: Remedial Action Plan, Allegheny Rodney Facility. Innovative Engineering Solutions, Inc., Norwood, MA. August 2002.

Notes:

Intermediate EMEG (adult) = Environmental Media Evaluation Guide for adults (i.e., for exposures b/t 14 days and 1 year).

Intermediate EMEG (child) = Environmental Media Evaluation Guide for children (i.e., for exposures b/t 14 days and 1 year and considers vulnerabilities of children when it comes to environmental exposures).

RMEGs (adult/child) = Reference Dose Media Evaluation Guides (an estimate of a daily exposure to the general public, including sensitive subgroups, that is likely to be without appreciable risk of deleterious effects during a specified duration of exposure).

CREG = Cancer Risk Evaluation Guide for 1×10^{-6} excess cancer risk

Chronic EMEGs (adult/child) = Environmental Media Evaluation Guides (i.e., for adult or childhood exposures mirroring greater than 1 year)

LTHA = Lifetime Health Advisory for Drinking Water (EPA)

NA = Not Available

* = Massachusetts Drinking Water Guideline

TABLE 3
Summary of Excavated Soil Sample Analytical Results
Rodney Metals, New Bedford, Massachusetts

COMPOUND	SAMPLING LOCATION	MAXIMUM DETECTED CONCENTRATION (ppm)	SOIL BACKGROUND CONCENTRATIONS* (ppm)	SOIL COMPARISON VALUE (ppm)
Total Petroleum Hydrocarbons (TPH)				
TPH	112194-03, Stockpile	34,000		Method 1 S-3/GW-2 standard = 5,000
Metals (which include lead and chromium)				
Chromium (total)	112194-01, Stockpile	6.5	52	Method 1 S-3/GW-2 standard = 5,000
Lead	112194-01, Stockpile	5	17	Method 1 S-3/GW-2 standard = 600

Data Sources:

Innovative Engineering Solutions, Inc. 2002. Phase II: Comprehensive Site Assessment and Phase II: Remedial Action Plan, Allegheny Rodney Facility. Innovative Engineering Solutions, Inc., Norwood, MA. August 2002.

Agency for Toxic Substances and Disease Registry (ATSDR). 1993. ATSDR Public Health Assessment Guidance Manual. Atlanta: U.S. Department of Health and Human Services.

TABLE 4
Summary of Surface Water Sample Analytical Results
Rodney Metals, New Bedford, Massachusetts

COMPOUND	SAMPLING LOCATION	MAXIMUM DETECTED CONCENTRATION (ppm)	DRINKING WATER COMPARISON VALUE	MAXIMUM CONTAMINANT LEVEL (MCL) (EPA) (ppb)
Volatile Organic Compounds (VOCs)				
1,1,1-Trichloroethane (1,1,1-TCA)	SW-03 (seawall low tide) (East of East Rodney French Blvd.)	12	LTHA = 200 ppb	200
Trichloroethylene (TCE)	SW-03 (seawall low tide)	2	NA	5
	SW-04 (endwall low tide)	2		

Data Source: Innovative Engineering Solutions, Inc. 2002. Phase II: Comprehensive Site Assessment and Phase II: Remedial Action Plan, Allegheny Rodney Facility. Innovative Engineering Solutions, Inc., Norwood, MA. August 2002.

TABLE 5

Cancer Incidence in CT 6528 : 1982-1998

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	14	16.0	87	48 -- 147	9	11.9	76	34 -- 143	5	4.1	122	39 -- 286
Breast	56	57.5	97	74 -- 126	1	0.5	NC	NC -- NC	55	57.0	96	73 -- 126
Kidney	12	8.7	139	71 -- 242	8	5.4	149	64 -- 294	4	3.3	NC	NC -- NC
Leukemia	12	7.0	171	88 -- 299	4	4.1	NC	NC -- NC	8	2.9	272	* 117 -- 537
Liver	2	2.0	NC	NC -- NC	1	1.5	NC	NC -- NC	1	0.5	NC	NC -- NC
Lung	41	58.1	71	* 51 -- 96	31	36.0	86	59 -- 122	10	22.2	45	* 22 -- 83
NHL	10	12.9	77	37 -- 142	5	6.8	74	24 -- 172	5	6.1	82	26 -- 191
Pancreas	10	8.4	119	57 -- 220	5	4.1	121	39 -- 282	5	4.2	118	38 -- 276

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry. Bureau of Health Statistics, Research & Evaluation, Massachusetts Department of Public Health.

TABLE 6

Cancer Incidence in CT 6528 : 1982-1986

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	4.8	NC	NC -- NC	2	3.6	NC	NC -- NC	2	1.2	NC	NC -- NC
Breast	15	14.8	101	57 -- 167	1	0.1	NC	NC -- NC	14	14.7	95	52 -- 160
Kidney	2	1.9	NC	NC -- NC	1	1.2	NC	NC -- NC	1	0.7	NC	NC -- NC
Leukemia	4	1.9	NC	NC -- NC	1	1.1	NC	NC -- NC	3	0.8	NC	NC -- NC
Liver	0	0.4	NC	NC -- NC	0	0.3	NC	NC -- NC	0	0.1	NC	NC -- NC
Lung	10	15.6	64	31 -- 118	9	10.5	86	39 -- 163	1	5.2	NC	NC -- NC
NHL	1	3.1	NC	NC -- NC	1	1.6	NC	NC -- NC	0	1.4	NC	NC -- NC
Pancreas	3	2.4	NC	NC -- NC	1	1.2	NC	NC -- NC	2	1.2	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry. Bureau of Health Statistics, Research & Evaluation, Massachusetts Department of Public Health.

TABLE 7

Cancer Incidence in CT 6528 : 1987-1994

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	6	7.3	82	30 -- 178	5	5.5	91	29 -- 212	1	1.8	NC	NC -- NC
Breast	24	27.9	86	55 -- 128	0	0.2	NC	NC -- NC	24	27.7	87	55 -- 129
Kidney	5	4.4	114	37 -- 265	2	2.8	NC	NC -- NC	3	1.6	NC	NC -- NC
Leukemia	5	3.1	160	52 -- 373	3	1.8	NC	NC -- NC	2	1.3	NC	NC -- NC
Liver	1	0.9	NC	NC -- NC	1	0.7	NC	NC -- NC	0	0.2	NC	NC -- NC
Lung	21	27.7	76	47 -- 116	14	17.1	82	45 -- 138	7	10.6	66	26 -- 136
NHL	7	6.3	112	45 -- 230	2	3.3	NC	NC -- NC	5	3.0	169	54 -- 394
Pancreas	3	3.8	NC	NC -- NC	3	1.9	NC	NC -- NC	0	1.9	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry. Bureau of Health Statistics, Research & Evaluation, Massachusetts Department of Public Health.

TABLE 8

Cancer Incidence in CT 6528 : 1995-1998

Cancer Type	Total				Males				Females			
	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI	Obs	Exp	SIR	95% CI
Bladder	4	3.5	NC	NC -- NC	2	2.6	NC	NC -- NC	2	0.9	NC	NC -- NC
Breast	17	13.7	124	72 -- 199	0	0.1	NC	NC -- NC	17	13.5	126	73 -- 202
Kidney	5	2.1	233	75 -- 545	5	1.3	380	* 123 -- 887	0	0.8	NC	NC -- NC
Leukemia	3	1.8	NC	NC -- NC	0	1.0	NC	NC -- NC	3	0.8	NC	NC -- NC
Liver	1	0.6	NC	NC -- NC	0	0.4	NC	NC -- NC	1	0.2	NC	NC -- NC
Lung	10	13.6	73	35 -- 135	8	7.7	104	45 -- 206	2	5.9	NC	NC -- NC
NHL	2	3.3	NC	NC -- NC	2	1.7	NC	NC -- NC	0	1.6	NC	NC -- NC
Pancreas	4	2.0	NC	NC -- NC	1	1.0	NC	NC -- NC	3	1.0	NC	NC -- NC

Note: SIRs are calculated based on the exact number of expected cases.
Expected number of cases presented are rounded to the nearest tenth.
SIRs and 95% CI are not calculated when observed number of cases < 5.

Obs = Observed number of cases
Exp = Expected number of cases
SIR = Standardized Incidence Ratio
NHL = Non-Hodgkin's Lymphoma
95% CI = 95% Confidence Interval
NC = Not calculated
* = Statistical significance

Data Source: Massachusetts Cancer Registry. Bureau of Health Statistics, Research & Evaluation, Massachusetts Department of Public Health.

PREPARER

This document was prepared by the Bureau of Environmental Health Assessment of the Massachusetts Department of Public Health. If you have any questions about this document, please contact Suzanne K. Condon, Assistant Commissioner of BEHA/MDPH at 250 Washington Street, 7th Floor, Boston, MA 02108.

Appendix A

Risk Factor Information for Selected Cancer Types

Bladder Cancer

The American Cancer Society estimates that bladder cancer will affect 56,500 people in the U.S. in 2002, accounting for 7% of all cancers diagnosed in the United States among men and 2% among women. In Massachusetts, bladder cancer accounts for approximately 5% of all cancers diagnosed among males and females combined (ACS, 2002). Males are three times more likely to develop bladder cancer than females and whites are two times more likely to develop this disease than blacks. The risk of bladder cancer increases with age and the mean age at diagnosis is 68-69 years (ACS, 2000).

The greatest risk factor for bladder cancer is cigarette smoking. Smokers are more than twice as likely to develop bladder cancer compared to nonsmokers (ACS, 2000). The risk of developing bladder cancer increases with the number of packs smoked per day and with duration of smoking. Further, the risk of bladder cancer may be higher in women than in men who smoke comparable numbers of cigarettes (Castelao et al., 2001). Approximately 25-60% of all bladder cancers can be attributed to tobacco use (Johansson and Cohen, 1997). Smoking cessation has been found to reduce the risk of developing bladder cancer by 30% to 60% (Silverman et al., 1996).

Studies have also revealed a number of occupations that are associated with bladder cancer. In fact, exposures to chemicals in the workplace account for an estimated 20-25% of all bladder cancers diagnosed among men in the U.S. (Johansson and Cohen, 1997). Occupational exposure to aromatic amines, such as benzidine and 2-naphthylamine, increases the risk of bladder cancer (ACS, 2000). These chemicals were common in the dye industry in the past. A higher risk of bladder cancer has also been observed among aromatic amine manufacturing workers as well as among workers in the rubber, leather, textiles, printing, and paint products industries (ACS, 2000; Silverman et al., 1996). The development of new chemicals, changed worker exposures, and the elimination of many known bladder carcinogens in the workplace have caused shifts in those occupations considered to be high risk. For example, risks among dye, rubber, and leather workers have declined over time, while other occupations such as motor vehicle operation (e.g., drivers of trucks, buses, and taxis) and the aluminum industry have emerged as potential high-risk occupations (Silverman et al., 1996). However, specific occupational exposures in these occupations have not been confirmed and study findings are not consistent. Further, the risk of bladder cancer from occupational exposures may be increased among smokers (ACS, 2000).

Dietary factors such as consumption of fried foods as well as foods high in fat and cholesterol have been found to be associated with increased bladder cancer risk (Silverman et al., 1996). Use of the Chinese herb, *Aristocholia fangchi,* found in some dietary supplements, has also been linked with bladder cancer (ACS, 2000). Use of some anti-cancer drugs (e.g., cyclophosphamide and chlornaphazine), use of phenacetin, and infection with *Shistosoma haematobium* (a parasite found in Africa) are thought to be associated with the development of bladder cancer, however, not all epidemiological studies have produced convincing findings (Silverman et al., 1996).

Other risk factors for bladder cancer include a personal history of bladder cancer, certain rare birth defects involving the bladder, and exposure to ionizing radiation (ACS, 2000; Silverman et al., 1996). Exposure to chlorinated by-products in drinking water has also been suggested to increase bladder cancer risk, however, a recent population-based study found that an association was present only among smokers (Cantor et al., 1998).

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Breast Cancer

Breast cancer is the most frequently diagnosed cancer among women in both the United States and in Massachusetts. According to the North American Association of Central Cancer Registries, female breast cancer incidence in Massachusetts is the fifth highest among all states (Chen et al, 2000). Although during the 1980s breast cancer in the U.S. increased by about 4% per year, the incidence has leveled off to about 110.6 cases per 100,000 (ACS 2000). A similar trend occurred in Massachusetts and there was even a slight decrease in incidence (1%) between 1993 and 1997 (MCR 2000).

In the year 2002, approximately 203,500 women in the U.S. will be diagnosed with breast cancer (ACS 2002). Worldwide, female breast cancer incidence has increased, mainly among women in older age groups whose proportion of the population continues to increase as well (van Dijck, 1997). A woman's risk for developing breast cancer can change over time due to many factors, some of which are dependent upon the well-established risk factors for breast cancer. These include increased age, an early age at menarche (menstruation) and/or late age at menopause, late age at first full-term pregnancy, family history of breast cancer, and high levels of estrogen. Other risk factors that may contribute to a woman's risk include benign breast disease and lifestyle factors such as diet, body weight, lack of physical activity, consumption of alcohol, and exposure to cigarette smoke. Data on whether one's risk may be affected by exposure to environmental chemicals or radiation remains inconclusive. However, studies are continuing to investigate these factors and their relationship to breast cancer.

Family history of breast cancer does affect one's risk for developing the disease. Epidemiological studies have found that females who have a first-degree relative with premenopausal breast cancer experience a 3-fold greater risk. However, no increase in risk has been found for females with a first degree relative with postmenopausal breast cancer. If women have a first-degree relative with bilateral breast cancer (cancer in both breasts) at any age then their risk increases five-fold. Moreover, if a woman has a mother, sister or daughter with bilateral premenopausal breast cancer, their risk increases nine fold. (Broeders and Verbeek, 1997). In addition, twins have a higher risk of breast cancer compared to non-twins (Weiss et al, 1997).

A personal history of benign breast disease is also associated with development of invasive breast cancer. Chronic cystic or fibrocystic disease is the most commonly diagnosed benign breast disease. Women with cystic breast disease experience a 2-3 fold increase in risk for breast cancer (Henderson et al, 1996).

According to recent studies, approximately 10% of breast cancers can be attributed to inherited mutations in breast cancer related genes. Most of these mutations occur in the BRCA1 and BRCA2 genes. Approximately 50% to 60% of women who inherit BRCA1 or BRCA2 gene mutations will develop breast cancer by the age of 70 (ACS 2001).

Cumulative exposure of the breast tissue to estrogen and progesterone hormones may be one of the greatest contributors to risk for breast cancer (Henderson et al, 1996). Researchers suspect that early exposures to a high level of estrogen, even during fetal development, may add to one's risk of developing breast cancer later in life. Other studies have found that factors associated with increased levels of estrogen (i.e., neonatal jaundice, severe prematurity, and being a fraternal twin) may contribute to an elevated risk of developing breast cancer (Ekbom et al, 1997). Conversely, studies have revealed that women whose mothers experienced toxemia during pregnancy (a condition associated with low levels of estrogen) had a significantly reduced risk of developing

breast cancer. Use of estrogen replacement therapy is another factor associated with increased hormone levels and it has been found to confer a modest (less than two-fold) elevation in risk when used for 10-15 years or longer (Kelsey, 1993). Similarly, more recent use of oral contraceptives or use for 12 years or longer seems to confer a modest increase in risk for bilateral breast cancer in premenopausal women (Ursin et al, 1998).

Cumulative lifetime exposure to estrogen may also be increased by certain reproductive events during one's life. Women who experience menarche at an early age (before age 12) have a 20% increase in risk compared to women who experience menarche at 14 years of age or older (Broeders and Verbeek, 1997; Harris et al, 1992). Women who experience menopause at a later age (after the age of 50) have a slightly elevated risk for developing the disease (ACS 2001). Furthermore, the increased cumulative exposure from the combined effect of early menarche and late menopause has been associated with elevated risk (Lipworth, 1995). In fact, women who have been actively menstruating for 40 or more years are thought to have twice the risk of developing breast cancer than women with 30 years or less of menstrual activity (Henderson et al, 1996). Other reproductive events have also shown a linear association with risk for breast cancer (Wohlfahrt, 2001). Specifically, women who gave birth for the first time before age 18 experience one-third the risk of women who have carried their first full-term pregnancy after age 30 (Boyle et al, 1988). The protective effect of earlier first full-term pregnancy appears to result from the reduced effect of circulating hormones on breast tissue after pregnancy (Kelsey, 1993).

Diet, and particularly fat intake, is another factor suggested to increase a woman's risk for breast cancer. Currently, a hypothesis exists that the type of fat in a woman's diet may be more important than her total fat intake (ACS 1998; Wynder et al, 1997). Monounsaturated fats (olive oil and canola oil) are associated with lower risk while polyunsaturated (corn oil, tub margarine) and saturated fats (from animal sources) are linked to an elevated risk. However, when factoring in a woman's weight with her dietary intake, the effect on risk becomes less clear (ACS 1998). Many studies indicate that a heavy body weight elevates the risk for breast cancer in postmenopausal women (Kelsey, 1993), probably due to fat tissue as the principal source of estrogen after menopause (McTiernan, 1997). Therefore, regular physical activity and a reduced body weight may decrease one's exposure to the hormones believed to play an important role in increasing breast cancer risk (Thune et al, 1997).

Aside from diet, regular alcohol consumption has also been associated with increased risk for breast cancer (Swanson et al, 1996; ACS 2001). Women who consumed one alcoholic beverage per day experienced a slight increase in risk (approximately 10%) compared to non-drinkers, however those who consumed 2 to 5 drinks per day experienced a 1.5 times increased risk (Ellison et al., 2001; ACS 2001). Despite this association, the effects of alcohol on estrogen metabolism have not been fully investigated (Swanson et al, 1996).

To date, no specific environmental factor, other than ionizing radiation, has been identified as a cause of breast cancer. The role of cigarette smoking in the development of breast cancer is unclear. Some studies suggest a relationship between passive smoking and increased risk for breast cancer; however, confirming this relationship has been difficult due to the lack of consistent results from studies investigating first-hand smoke exposure (Laden and Hunter, 1998).

Studies on exposure to high doses of ionizing radiation demonstrate a strong association with breast cancer risk. These studies have been conducted in atomic bomb survivors from Japan as well as patients that have been subjected to radiotherapy in treatments for other conditions (i.e., Hodgkin's Disease, non-Hodgkin's Lymphoma, tuberculosis, post-partum mastitis, and cervical

cancer) (ACS 2001). However, it has not been shown that radiation exposures experienced by the general public or people living in areas of high radiation levels, from industrial accidents or nuclear activities, are related to an increase in breast cancer risk (Laden and Hunter, 1998). Investigations of electromagnetic field exposures in relation to breast cancer have been inconclusive as well.

Occupational exposures associated with increased risk for breast cancer have not been clearly identified. Experimental data suggests that exposure to certain organic solvents and other chemicals (e.g., benzene, trichloropropane, vinyl chloride, polycyclic aromatic hydrocarbons (PAHs)) causes the formation of breast tumors in animals and thus may contribute to such tumors in humans (Goldberg and Labreche, 1996). Particularly, a significantly elevated risk for breast cancer was found for young women employed in solvent-using industries (Hansen, 1999). Although risk for premenopausal breast cancer may be elevated in studies on the occupational exposure to a combination of chemicals, including benzene and PAHs, other studies on cigarette smoke (a source of both chemicals) and breast cancer have not shown an associated risk (Petralia et al, 1999). Hence, although study findings have yielded conflicting results, evidence does exist to warrant further investigation into the associations.

Other occupational and environmental exposures have been suggested to confer an increased risk for breast cancer in women, such as exposure to polychlorinated biphenyls (PCBs), chlorinated hydrocarbon pesticides (DDT and DDE), and other endocrine-disrupting chemicals. Because these compounds affect the body's estrogen production and metabolism, they can contribute to the development and growth of breast tumors (Davis et al, 1997; Holford et al, 2000; Laden and Hunter, 1998). However, studies on this association have yielded inconsistent results and follow-up studies are ongoing to further investigate any causal relationship (Safe, 2000).

When considering a possible relationship between any exposure and the development of cancer, it is important to consider the latency period. Latency refers to the time between exposure to a causative factor and the development of the disease outcome, in this case breast cancer. It has been reported that there is an 8 to 15 year latency period for breast cancer (Petralia 1999; Aschengrau 1998; Lewis-Michl 1996). That means that if an environmental exposure were related to breast cancer, it may take 8 to 15 years after exposure to a causative factor for breast cancer to develop.

Socioeconomic differences in breast cancer incidence may be a result of current screening participation rates. Currently, women of higher socioeconomic status (SES) have higher screening rates, which may result in more of the cases being detected in these women. However, women of higher SES may also have an increased risk for developing the disease due to different reproductive patterns (i.e., parity, age at first full-term birth, and age at menarche). Although women of lower SES show lower incidence rates of breast cancer in number, their cancers tend to be diagnosed at a later stage (Segnan, 1997). Hence, rates for their cancers may appear lower due to the lack of screening participation rather than a decreased risk for the disease. Moreover, it is likely that SES is not in itself the associated risk factor for breast cancer. Rather, SES probably represents different patterns of reproductive choices, occupational backgrounds, environmental exposures, and lifestyle factors (i.e., diet, physical activity, cultural practices) (Henderson et al, 1996).

Despite the vast number of studies on the causation of breast cancer, known factors are estimated to account for less than half of breast cancers in the general population (Madigan et al, 1995). Researchers are continuing to examine potential risks for developing breast cancer, especially environmental factors.

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Kidney cancer

Kidney cancer involves a number of tumor types located in various areas of the kidney and renal system. Renal cell cancer (which affects the main area of the kidney) accounts for over 90% of all malignant kidney tumors (ACS, 2001). The American Cancer Society estimates that there will be approximately 31,800 cases of kidney and upper urinary tract cancer, resulting in more than 11,600 deaths in 2002 (ACS, 2002). The incidence and mortality from kidney cancer is higher in urban areas, which may be due to increased access to diagnostic services and other factors such as smoking. Kidney cancer is twice as common in males as it is in females and the incidence most often occurs in the fifth and sixth decades of life (50-70 year age group) (ACS, 2001). The gender distribution of this disease may be attributed to the fact that men are more likely to smoke and are more likely to be exposed to potentially carcinogenic chemicals at work.

Since 1970, U.S. incidence rates for renal cell cancer have risen between 2 and 4% annually among the four major race and gender groups (i.e., white males, white females, black males, and black females) (Chow et al., 1999; McLaughlin et al., 1996). Rapid increases in incidence among blacks as compared to among whites have resulted in an excess of the disease among blacks; age-adjusted incidence rates between 1975 and 1995 for white men, white women, black men, and black women were 9.6, 4.4, 11.1, and 4.9 per 100,000 person-years, respectively (Chow et al., 1999). Rising incidence rates may be partially due to the increased availability of screening for kidney cancer.

The etiology of kidney cancer is not fully understood. However, a number of environmental, hormonal, cellular, and genetic factors have been studied as possible causal factors in the development of renal cell carcinoma. Cigarette smoking is the most important known risk factor for renal cell cancer. Smoking increases the risk of developing renal cell cancer by 30% to 100% (ACS, 2001). In both males and females, a statistically significant dose-response relationship between smoking and this cancer has been observed. Approximately one-third of renal cell cancers in men and one-quarter of those in women may be caused by cigarette smoking (ACS, 2001).

Virtually every study that has examined body weight and renal cell cancer has observed a positive association. Some studies suggest that obesity is a factor in 20% of people who develop kidney cancer (ACS, 2001). This is especially true among women and researchers suspect that this may be related to changes in certain hormones, such as estrogen in women (ACS, 2001; McLaughlin et al., 1996). A diet high in protein (meat, animal fats, milk products, margarine and oils) has been implicated in epidemiological studies as a risk factor for renal cell carcinoma (ACS, 2001; McLaughlin et al., 1996). Consumption of adequate amounts of fruits and vegetables lowers the risk of renal cell cancer. In addition, use of diuretics and antihypertensive medications are associated with increased risk of renal cell carcinoma. However, hypertension has also been linked to kidney cancer and it is not clear whether the disease or the medications used to treat them is the cause (ACS, 2001). Long-term use of pain relievers such as phenacetin (and possibly acetaminophen and aspirin) increases the risk for cancer of the renal pelvis and renal cell carcinoma (ACS, 2001).

Certain medical conditions that affect the kidneys have also been shown to increase kidney cancer risk. There is an increased incidence of renal carcinoma in patients with end-stage renal disease who develop acquired cystic disease of the kidney. This phenomenon is seen among patients on long-term dialysis for renal failure (Linehan et al., 1997). In addition, an association has been established between the incidence of von Hippel-Lindau disease and certain other inherited conditions in families and renal cell carcinoma, suggesting that genetic and hereditary

risk factors may be important in the development of kidney cancer (ACS, 2001; McLaughlin et al., 1996).

Environmental and occupational factors have also been associated with the development of kidney cancer. Some studies have shown an increased incidence of this cancer type among leather tanners, shoe workers, and workers exposed to asbestos. Exposure to cadmium is associated with an increased incidence of kidney cancer, particularly in men who smoke (ACS, 2001; Linehan et al,. 1997). In addition, workplace exposure to organic solvents, particularly trichloroethylene, may increase the risk of this cancer (ACS, 2001). Although occupational exposure to petroleum, tar, and pitch products has been implicated in the development of kidney cancer, most studies of oil refinery workers and petroleum products distribution workers have not identified a definitive relationship between gasoline exposure and renal cancer (Linehan et al., 1997; McLaughlin et al., 1996).

Wilms' tumor is the most common type of kidney cancer affecting children and accounts for approximately 5% to 6% of all kidney cancers and about 6% of all childhood cancers. This cancer is more common among African Americans than other races and among females than males. Wilms' tumor most often occurs in children under the age of 5 years. The causes of Wilms' tumor are not known, but certain birth defect syndromes and other genetic risk factors (such as family history or genetic mutations) are connected with this cancer. However, most children who develop Wilms' tumor do not have any known birth defects or inherited gene changes. No environmental risk factors, either before or after a child's birth, have been shown to be associated with the development of Wilms' tumor (ACS, 1999).

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Leukemia

Leukemia is the general term that includes a group of different cancers that occur in the blood forming organs and result in the formation of abnormal amounts and types of white blood cells in the blood and bone marrow. Individuals with leukemia generally maintain abnormally high amounts of leukocytes or white blood cells in their blood. This condition results in an individual's inability to maintain certain body functions, particularly a person's ability to combat infection.

In 2002, leukemia is expected to affect approximately 30,800 individuals (17,600 males and 13,200 females) in the United States, resulting in 21,700 deaths. In Massachusetts, approximately 700 individuals will be diagnosed with the disease in 2002, representing more than 2% of all cancer diagnoses. There are four major types of leukemia: acute lymphoid leukemia (ALL), acute myeloid leukemia (AML), chronic lymphoid leukemia (CLL), and chronic myeloid leukemia (CML). There are also a few rare types, such as hairy cell leukemia. In adults, the most common types are AML and CLL. Leukemia is the most common type of childhood cancer, accounting for more than 30% of all cancers diagnosed in children. The majority of these cases are of the ALL type (ACS 2002).

While ALL occurs predominantly among children (peaking between ages 2 and 3 years), an elevation in incidence is also seen among older individuals. The increase in incidence among older individuals begins at approximately 40-50 years of age, peaking at about age 85 (Linet and Cartwright, 1996). ALL is more common among whites than African Americans and among males than females (Weinstein and Tarbell, 1997). Exposure to high-dose radiation (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents) is a known environmental risk factor associated with the development of ALL (Scheinberg et al., 1997). Significant radiation exposure (e.g., diagnostic x-rays) before birth may carry up to a 5-fold increased risk of developing ALL (ACS 2000b). However, few studies report an increased risk of leukemia associated with residing in proximity to nuclear plants or occupational exposure to low-dose radiation (Linet and Cartwright, 1996; Scheinberg et al., 1997). It is unclear whether exposure to electromagnetic fields (EMF) plays a role in the development of ALL, however, most studies to date have found little or no risk (ACS 2000b).

Few other risk factors for ALL have been identified. There is evidence that genetics may play an important role in the development of this leukemia type. Studies indicate that siblings of twins who develop leukemia are at an increased risk of developing the disease. Children with Down's syndrome are 10 to 20 times more likely to develop acute leukemia (Weinstein and Tarbell, 1997). In addition, other genetic diseases, such as Li-Fraumeni syndrome and Klinefelter's syndrome, are associated with an increased risk of developing leukemia. Patients receiving medication that suppresses the immune system (e.g., organ transplant patients) may be more likely to develop ALL (ACS 2000b). ALL has not been definitively linked to chemical exposure, however, childhood ALL may be associated with maternal occupational exposure to pesticides during pregnancy (Infante-Rivard et al., 1999). Certain rare types of adult ALL are caused by human T-cell leukemia/lymphoma virus-I (HTLV-I) (ACS, 2000a). Some reports have linked other viruses with various types of leukemia, including Epstein-Barr virus and hepatitis B virus. Still others propose that leukemia may develop as a response to viral infection. However, no specific virus has been identified as related to ALL (Linet and Cartwright, 1996). Recent reports also suggest an infectious etiology for some childhood ALL cases, although a specific viral agent has not been identified and findings from studies exploring contact among children in day-care

do not support this hypothesis (Greaves MF, 1997; Kinlen and Balkwill, 2001; Rosenbaum et al., 2000).

Although AML can occur in children (usually during the first two years of life), AML is the most common leukemia among adults, with an average age at diagnosis of 65 years (ACS, 2000a and 2000b). This type of leukemia is more common among males than among females but affects African Americans and whites at similar rates (Scheinberg et al., 1997). High-dose radiation exposure (e.g., by survivors of atomic bomb blasts or nuclear reactor accidents), long-term occupational exposure to benzene, and exposure to certain chemotherapy drugs, especially alkylating agents (e.g., mechlorethamine, cyclophosphamide), have been associated with an increased risk of developing AML among both children and adults (ACS, 2000a and 2000b; Linet and Cartwright, 1996). The development of childhood AML is suspected to be related to parental exposure to pesticides and other chemicals, although findings are inconsistent (Linet and Cartwright, 1996). Recent studies have suggested a link between electromagnetic field (EMF) exposure (e.g., from power lines) and leukemia (Minder and Pfluger, 2001; Schuz et al., 2001). However, there is conflicting evidence regarding EMF exposure and leukemia and it is clear that most cases are not related to EMF (ACS, 2000a; Kleinerman et al., 2000).

Other possible risk factors related to the development of AML include cigarette smoking and genetic disorders. It is estimated that approximately one-fifth of cases of AML are caused by smoking (Scheinberg et al., 1997). Also, a small number of AML cases can be attributed to rare inherited disorders. These include Down's syndrome in children, Fanconi's anemia, Wiskott-Aldrich syndrome, Bloom's syndrome, Li-Fraumeni syndrome, and ataxia telangiectasia (ACS, 2000a and 2000b). Recently, scientists have suggested that a mutation in a gene responsible for the deactivation of certain toxic metabolites may have the ability to increase the risk of acute myeloid leukemia in adults. However, further research is necessary in order to confirm the findings of this study (Smith et al., 2001).

CLL is chiefly an adult disease; the average age at diagnosis is about 70 years (ACS 1999). Twice as many men as women are affected by this type of leukemia (Deisseroth et al., 1997). While genetics and diseases of the immune system have been suggested as playing a role in the development of CLL, high-dose radiation and benzene exposure have not (ACS, 1999; Weinstein and Tarbell, 1997). It is thought that individuals with a family history of CLL are two to four times as likely to develop the disease. Some studies have identified an increased risk of developing CLL (as well as ALL, AML, and CML) among farmers due to long-term exposure to herbicides and/or pesticides (Linet and Cartwright, 1996). In addition, many researchers believe that cigarette smoking plays a role in some chronic leukemias. The role of EMF in the development of chronic leukemia remains controversial (ACS, 1999). Although viruses have been implicated in the etiology of other leukemias, there is no evidence that viruses cause CLL (Deisseroth et al., 1997).

Of all the leukemias, CML is among the least understood. While this disease can occur at any age, CML is extremely rare in children (about 2% of leukemias in children) and the average age of diagnosis is 40 to 50 years (ACS 1999). Incidence rates are higher in males than in females, but unlike the other leukemia types, rates are higher in blacks than in whites in the U.S. (Linet and Cartwright, 1996). High-dose radiation exposure may increase the risk of developing CML (ACS, 1999). Finally, CML has been associated with chromosome abnormalities such as the Philadelphia chromosome (Weinstein and Tarbell, 1997).

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Liver Cancer

An estimated 16,600 people in the U.S. (11,000 men and 5,600 women) will be diagnosed with liver cancer in 2002, accounting for approximately 1% of all new cancers (ACS, 2002). Hepatocellular carcinoma (HCC) is the most common primary cancer of the liver, accounting for about 75% of all cases. Rarer forms of malignant liver cancer include cholangiocarcinomas, angiosarcomas, and hepatoblastomas in children. Although HCC is approximately ten times more common in developing countries in East and Southeast Asia and Africa, incidence is rapidly increasing in the United States (ACS, 2001). Rates of HCC in the U.S. have increased by 70% over the past two decades (Yu et al., 2000). Similar trends have been observed in Canada and Western Europe. The primary reason for the higher rates observed in recent years is the increase in hepatitis C virus infection, an important factor related to liver cancer (El-Serag, 2001; El-Serag and Mason, 2000). Men are at least two to three times more likely to develop liver cancer than women (Yu et al., 2000). Incidence rates are also higher among African Americans than whites. Although the risk of developing HCC increases with increasing age, the disease can occur in persons of any age (London and McGlynn, 1996).

Several important risk factors for liver cancer have been identified. Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) are the most significant risk factors for developing liver cancer (ACS, 2001). It is estimated that 80% of HCC cases worldwide can be attributed to HBV infection (Yu et al., 2000). However, HBV accounts for only about a quarter of the cases in the U.S. and infection with HCV plays a much larger role in the incidence of this cancer. HBV and HCV can be spread through intravenous drug use (e.g., the sharing of contaminated needles), unprotected sexual intercourse, and transfusion of and contact with unscreened blood and blood products. In addition, mothers who are infected with these viruses can pass them on to their children at birth or in early infancy (ACS, 2001).

Cirrhosis is also a major risk factor for the development of liver cancer. Cirrhosis is a progressive disease that causes inflammation and scar tissue to form on the liver, which can often lead to cancer. Researchers estimate that 60% to 80% of HCC cases are associated with cirrhosis. However, it is unclear if cirrhosis itself causes liver cancer or if the underlying causes of cirrhosis contribute to the development of this disease (Garr et al., 1997). Most liver cirrhosis in the U.S. occurs as a result of chronic alcohol abuse, but HBV and HCV are also major causes of cirrhosis (ACS, 2001). In addition, certain inherited metabolic diseases, such as hemochromatosis, which causes excess iron accumulation in the body, can lead to cirrhosis (ACS, 2001). Some studies have shown that people with hemochromatosis are at an increased risk of developing liver cancer (Fracanzani et al., 2001).

Epidemiological and environmental evidence indicates that exposure to certain chemicals and toxins can also contribute significantly to the development of liver cancer. For example, chronic consumption of alcoholic beverages has been associated with liver cancer (Wogan, 2000). As noted above, it is unclear if alcohol itself causes HCC or if underlying cirrhosis is the cause (London and McGlynn, 1996). However, it is clear that alcohol abuse can accelerate liver disease and may act as a co-carcinogen in the development of liver cancer (Ince and Wands, 1999). Long-term exposure to aflatoxin can also cause liver cancer. Aflatoxins are carcinogenic agents produced by a fungus found in tropical and subtropical regions. Individuals may be exposed to aflatoxins if they consume contaminated peanuts and other foods that have been stored under hot, humid conditions (Wogan, 2000). Vinyl chloride, a known human carcinogen used in the manufacturing of some plastics, and thorium dioxide, used in the past for certain x-

ray tests, are risk factors for a rare type of liver cancer called angiosarcoma (ACS, 2001; London and McGlynn, 1996). These chemicals may also increase the risk of HCC, but to a lesser degree. The impact of both thorium dioxide and vinyl chloride on the incidence of liver cancer was much greater in the past, since thorium dioxide has not been used for decades and exposure of workers to vinyl chloride is now strictly regulated in the U.S. (ACS, 2001). Drinking water contaminated with arsenic may increase the risk of liver cancer in some parts of the world (ACS, 2001; ATSDR, 2001).

The use of oral contraceptives by women may also be a risk factor in the development of liver cancer. However, most of the studies linking oral contraceptives and HCC involved types of oral contraceptives that are no longer used. There is some indication that the increased risk may be confined to oral contraceptives containing mestranol. It is not known if the newer oral contraceptives, which contain different types and doses of estrogen and different combinations of estrogen with other hormones, significantly increase the risk of HCC (ACS, 2001; London and McGlynn, 1996). Long-term anabolic steroid use may slightly increase the risk of HCC; however, a definitive relationship has not been established (ACS, 2001; London and McGlynn, 1996). Although many researchers believe that cigarette smoking plays a role in the development of liver cancer, the evidence for this is still inconclusive (Mizoue et al., 2000; London and McGlynn, 1996).

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Lung Cancer

Lung cancer generally arises in the epithelial tissue of the lung. Several different histologic or cell types of lung cancer have been observed. The various types of lung cancer occur in different regions of the lung and each type is associated with slightly different risk factors (Blot and Fraumeni 1996). The most common type of lung cancer in the United States today is adenocarcinoma which accounts for about 40% of all lung cancers (ACS, 2000). The greatest established risk factor for all types of lung cancer is cigarette smoking, followed by occupational and environmental exposures.

The incidence of lung cancer increases sharply with age peaking at about age 60 or 70. Lung cancer is very rare in people under the age of 40. The incidence is greater among men than women (probably because men are more likely to be smokers than women) and among blacks than whites (Blot and Fraumeni, 1996). The American Cancer Society estimates that lung cancer will be diagnosed in 169,400 people in the U.S. in 2002, accounting for about 13% of all cancers (ACS, 2002). Lung cancer is the leading cause of cancer death among both men and women; more people die of lung cancer than of colon, breast, and prostate cancers combined (ACS, 2000). In Massachusetts, incidence rates in 1997 were 76.7 per 100,000 and 49.2 per 100,000 for males and females, respectively (MCR, 2000). Nationwide, the incidence rate declined significantly in men during the 1990s, most likely as a result of decreased smoking rates over the past 30 years. Rates for women have continued to increase, but at a much slower pace and have begun to level off. This is because decreasing smoking patterns among women have lagged behind those of men (ACS, 2002). Trends in lung cancer incidence suggest that the disease has become increasingly associated with populations of lower socioeconomic status, since these individuals have higher rates of smoking than individuals of other groups (Blot and Fraumeni 1996).

More than 80% of all lung cancers are caused directly by smoking cigarettes and many of the rest are due to exposure to second hand smoke, or environmental tobacco smoke. The longer a person has been smoking and the higher the number of cigarettes smoked per day, the greater the risk of lung cancer. Smoking cessation decreases the elevated risk by about 50%, however, former smokers still carry a greater risk than those who have never smoked (ACS, 2000).

Workplace exposures have also been identified as playing important roles in the development of lung cancer. Occupational exposure to asbestos is an established risk factor for this disease; asbestos workers are about seven times more likely to die from lung cancer than the general population (ACS, 2000). Underground miners exposed to radon and uranium are at an increased risk for developing lung cancer (ACS, 2000; Samet and Eradze, 2000). Chemical workers, talc miners and millers, paper and pulp workers, carpenters, metal workers, butchers and meat packers, vineyard workers, carpenters and painters, and shipyard and railroad manufacture workers are some of the occupations associated with an increased risk of lung cancer (Blot and Fraumeni, 1996; Pohlablen et al., 2000). In addition to asbestos and radon, chemical compounds such as arsenic, chloromethyl ethers, chromium, vinyl chloride, nickel chromates, coal products, mustard gas, ionizing radiation, and fuels such as gasoline are also occupational risk factors for lung cancer (ACS, 2000; Blot and Fraumeni, 1996). Industrial sand workers exposed to crystalline silica are also at an increased risk for lung cancer (Rice et al., 2001; Steenland and Sanderson, 2001). Occupational exposure to the compounds noted above in conjunction with cigarette smoking dramatically increases the risk of developing lung cancer (Blot and Fraumeni, 1996).

As noted above, exposure to radon (a naturally occurring radioactive gas produced by the breakdown of radium and uranium) has been associated with increased risk of developing lung cancer among miners. Recently, a number of studies have demonstrated that exposure to elevated levels of residential radon may also increase lung cancer risk (Lubin and Boice, 1997; Kreienbrock et al., 2001; Tomasek et al., 2001). Epidemiological evidence suggests that radon may be the second leading cause of lung cancer after smoking (Samet and Eradze, 2000). However, actual lung cancer risk is determined by cumulative lifetime exposure to indoor radon. Therefore, normal patterns of residential mobility suggest that most people living in high-radon homes experience lifetime exposures equivalent to residing in homes with lower radon levels (Warner et al., 1996).

Tuberculosis and some types of pneumonia may increase the risk of lung cancer due to scarred lung tissue (ACS, 2000). In addition, people who have had lung cancer have a higher risk of developing another tumor. A family history of lung cancer may also slightly increase the risk, however, it is unclear whether this is due to inherited factors or environmental tobacco smoke (ACS, 2000).

Air pollution may increase the risk of developing lung cancer, however, this risk is much lower than that due to cigarette smoking (ACS, 2000).

Diet has also been implicated in the etiology of lung cancer, however, the exact relationship is unclear. Diets high in fruits and vegetables decrease lung cancer risk, but the reasons for this are unknown (Brownson et al., 1998). A recent study showed a positive association between total fat, monounsaturated fat, and saturated fat and lung cancer among males, however, this effect was not observed among women (Bandera et al., 1997).

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Non-Hodgkin's Lymphoma

Lymphomas are cancers involving the cells of the lymphatic system. The majority of lymphomas involve the lymph nodes and spleen but the disease may also affect other areas within the body. Non-Hodgkin's lymphoma (NHL) is a classification of all lymphomas except Hodgkin's disease. Thus NHL is a mixed group of diseases that is characterized by the malignant increase in specific cells of the immune system (B or T lymphocytes). B-cell lymphomas are more common than T-cell lymphomas, accounting for about 85% of all cases of NHL (ACS, 1998). The various types of NHL are thought to represent different diseases with different causes (Scherr and Mueller, 1996). NHL can occur at all ages, however, the median age at diagnosis is in the early 40s and the incidence of this disease generally increases with age. This disease is more common in men than in women and affects whites more often than African Americans or Asian Americans (ACS, 1998). The American Cancer Society estimates that approximately 53,900 Americans will be diagnosed with NHL in 2002, making it the sixth most common cancer in the U.S. among men and the fifth most common cancer in the U.S. among women, excluding non-melanoma skin cancers (ACS, 2002).

Overall, between 1973 and 1997, the incidence of NHL in the U.S. grew 81% (Garber, 2001), although during the 1990s, the rate of increase appears to have stabilized (ACS, 2002). In Massachusetts, the incidence of NHL increased 50% during 1982-1997 from 10.5 cases per 100,000 to 15.7 cases per 100,000 (MCR, 1997 and 2000). The increase in NHL incidence has been attributed to better diagnosis, greater exposure to causative agents, and, to a lesser extent, the increasing incidence of AIDS-related lymphomas (Devesa and Fears, 1992; Scherr and Mueller, 1996). Although the primary factors related to the development of NHL include conditions that suppress the immune system, viral infections, and certain occupational exposures, these factors are thought to account for only a portion of the increase observed in this cancer type (Scherr and Mueller, 1996). The observation that the rate of increase is declining for NHL may be attributed in part to increased use of antiretroviral therapy to slow HIV progression (Wingo et al., 1998).

NHL is more common among people who have abnormal or compromised immune systems, such as those with inherited diseases that suppress the immune system, individuals with autoimmune disorders, and people taking immunosuppressant drugs following organ transplants. Genetic predisposition (e.g., inherited immune deficiencies) only accounts for a small proportion of NHL cases (Scherr and Mueller, 1996). AIDS patients have a 100- to 300-fold higher risk for NHL than the general population (again, these cases account for only a minor part of overall NHL incidence) (Garber, 2001). NHL has also been reported to occur more frequently among individuals with conditions that require medical treatment resulting in suppression of the immune system, such as cancer chemotherapy. However, current evidence suggests that the development of NHL is related to suppression of the individual's immune system as a result of treatment, rather than the treatment itself (Scherr and Mueller, 1996).

Several viruses have been shown to play a role in the development of NHL. Among organ transplant recipients, suppression of the immune system required for acceptance of the transplant leads to a loss of control or the reactivation of viruses that have been dormant in the body (e.g., Epstein-Barr Virus [EBV] and herpesvirus infections). In addition, because cancer-causing viruses are known to cause lymphomas in various animals, it has been proposed that these types of viruses may also be associated with the development of NHL among humans without compromised immune systems. Infection with the human T-cell leukemia/lymphoma virus (HTLV-I) is known to

cause T-cell lymphoma among adults. However, this is a relatively rare infection and most likely contributes only a small amount to the total incidence of NHL (Scherr and Mueller, 1996). EBV infection is common among the general population and has been shown to play a role in the development of most cases of transplant and AIDS related NHL. The combination of immune system deficiencies and EBV infection may cause some people to develop NHL (ACS, 1998). Although viruses are causal factors for some subtypes of NHL, to date, studies have shown that the role of EBV in the development of NHL in the general population may not be large (Scherr and Mueller, 1996). Moreover, the high prevalence of EBV in the general population suggests that EBV may be only one of several factors in the development of this cancer.

Recent studies have found that a type of bacteria, *Helicobacter pylori*, a common cause of stomach ulcers, can also cause some lymphomas of the stomach (ACS, 1998). An important implication of this finding is that treatment with antibiotics could prevent some NHL of the stomach.

Some occupations have been associated with an increased risk of developing NHL, such as occupations related to chemicals or agriculture. Farmers, herbicide and pesticide applicators, and grain workers appear to have the most increased risk (Zahm, 1990 and 1993; Tatham et al., 1997). Studies conducted among agricultural workers have demonstrated increases in NHL among those using herbicides for more than 20 days per year and individuals who mix or apply herbicides. A greater incidence of NHL appears to be related specifically to exposure to the herbicide 2,4-dichlorophenoxyacetic acid (2,4-D) and organophosphate insecticides (Wigle et al., 1990; Zahm et al., 1990; Zahm et al., 1993). Further studies of exposure to these chemicals and NHL incidence have shown that the increased risk is attributed to a specific impurity, 2,3,7,8-tetrachlorodibenzo-p-dioxin or 2,3,7,8-TCDD, present in these herbicides. However, reports of accidental industrial exposures to TCDD alone have not demonstrated an increased risk of NHL (Scherr and Mueller, 1996). An elevated risk for NHL development has also been noted among fence workers, orchard workers, and meat workers. High-dose exposure to benzene has been associated with NHL (ACS, 1998), however, a recent international cohort study indicated that petroleum workers exposed to benzene were not at an increased risk of NHL (Wong and Raabe, 2000).

In addition, epidemiological studies of long-term users of permanent hair coloring products have suggested an increased incidence of NHL (Zahm et al., 1992; Scherr and Mueller, 1996). However, a recent population based study found no association between the use of hair color products and an increased risk of developing NHL. The researchers further stated that results from this study and previous studies, including experimental animal studies, provide little convincing evidence linking NHL with normal use of hair dye (Holly et al., 1998).

Although radiation (e.g., nuclear explosions or radioactive fallout from reactor accidents) has been implicated in the development of some cancers, including NHL (ACS, 1998), there is little evidence for an increased risk of lymphoma due to radiation (Scherr and Mueller, 1996).

Recent studies have suggested that contamination of drinking water with nitrate may be associated with an increased risk of NHL (Ward et al., 1996). Nitrate forms N-nitroso compounds which are known carcinogens and can be found in smoked or salt-dried fish, bacon, sausages, other cured meats, beer, pickled vegetables, and mushrooms.

Smoking has also been suggested to increase the risk of NHL. A study that evaluated the history of tobacco use and deaths from NHL determined that people who had ever smoked had a two-fold increase of dying from NHL as compared to those who never smoked. Further, a four-fold increase was found among the heaviest smokers (Linet et al., 1992). In addition, a more recent study that

primarily examined occupation and NHL risk found a significant association with high levels of cigarette smoking and all NHL types (Tatham et al., 1997). However, a recent review of 5 cohort studies and 14 case-control studies concludes that results of epidemiological studies have been inconsistent and that smoking has not been determined to be a definitive risk factor in the development of NHL (Peach and Barnett, 2000).

A recent Danish study has linked the use of tricyclic and tetracyclic antidepressants to NHL, however, more research is needed on this possible association (Dalton et al., 2000).

Although NHL is associated with a number of risk factors, the causes of this disease remain unknown. Most patients with NHL do not have any known risk factors (ACS, 1998).

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Pancreatic Cancer

The American Cancer Society estimates that approximately 30,300 people in the U.S. (14,700 men and 15,600 women) will develop pancreatic cancer in 2002. This disease accounts for approximately 2% of all new cases of cancer in both men and women, but between 5% and 6% of all cancer deaths (ACS, 2002). This discrepancy has been attributed to detection of pancreatic cancer at an advanced stage and the short median survival time for this cancer of approximately three months. Between 1920 and 1965, mortality from this disease increased nearly 200% from 2.9 to 8.2 per 100,000 people. These increases are believed to be due, in part, to improved diagnosis during this time period (Anderson et al., 1996). However, over the past 25 years, incidence rates have declined slowly but consistently in men and a slight decline in rates among women has been observed since the mid-1980s. Further, since about 1975, men have experienced a slight decrease in mortality from pancreatic cancer, although rates among women have not dropped (ACS, 2002). The risk of developing pancreatic cancer increases with age and the majority of cases occur between age 60 and 80. Men are approximately 30% more likely to develop pancreatic cancer than are women (ACS, 2000).

Very little is known about what causes pancreatic cancer and how to prevent it. However, a number of risk factors have been identified. Besides age, the most consistent and only established risk factor for pancreatic cancer is cigarette smoking. According to the American Cancer Society, approximately 30% of all pancreatic cancer cases are thought to result directly from cigarette smoking (ACS, 2000). Studies have estimated that the risk of pancreatic cancer is two to six times greater in heavy smokers than in non-smokers (Anderson et al., 1996).

Certain medical conditions, such as chronic pancreatitis, diabetes mellitus, and cirrhosis, have been associated with pancreatic cancer, but the reasons for these associations are largely unknown (ACS, 2000). More recently, a possible role for the bacteria *Helicobacter pylori,* which causes ulcers and some gastric cancers, has been suggested in the development of pancreatic cancer (Stolzenberg-Solomon et al., 2001).

There is also some evidence to suggest that certain dietary factors may be related to the development of pancreatic cancer. Increased risks of pancreatic cancer may be associated with animal protein and fat consumption as evidenced by higher rates of this cancer in countries whose populations eat a diet high in fat (ACS, 2002). Decreased risks for the disease are usually associated with fruit and vegetable consumption (ACS, 2000). Obesity is also a risk factor for pancreatic cancer (ACS, 2000). Although older studies suggested that coffee and alcohol consumption may be risk factors, more recent studies do not support this association (Michaud et al., 2001).

Numerous occupations have been investigated for their potential role in the development of pancreatic cancer, but studies have not produced consistent results. Heavy exposure to certain pesticides (including DDT and its derivatives) may increase the risk of pancreatic cancer (ACS, 2000; Ji et al., 2000; Porta et al., 1999). Exposure to certain dyes and certain chemicals related to gasoline, in addition to asbestos and ionizing radiation, have also been associated with the development of pancreatic cancer in some studies, however, other studies have found no link between these agents and pancreatic cancer (ACS, 2000; Anderson et al., 1996). A recent evaluation of data from several studies has implicated organic solvents (e.g., chlorinated hydrocarbons and polycyclic aromatic hydrocarbons), nickel compounds, and chromium compounds in the development of pancreatic cancer, but further studies are needed to

corroborate this claim (Ojajarvi et al., 2000). Although occupational exposures may have played a role in the incidence of this cancer in the past, currently most newly diagnosed patients with pancreatic cancer do not have evidence of a specific chemical exposure or relevant occupational history (Evans et al., 1997).

Finally, pancreatic cancer seems to run in some families. According to the American Cancer Society, an inherited tendency to develop pancreatic cancer may account for approximately 5% to 10% of cases (ACS, 2000). Pancreatic cancer has been observed in both familial clusterings among siblings as well as in individuals of consecutive generations (Anderson et al., 1996).

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Appendix B

ATSDR Glossary of Environmental Health Terms

ATSDR Glossary of Environmental Health Terms

The Agency for Toxic Substances and Disease Registry (ATSDR) is a federal public health agency with headquarters in Atlanta, Georgia, and 10 regional offices in the United States. ATSDR's mission is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and diseases related to toxic substances. ATSDR is not a regulatory agency, unlike the U.S. Environmental Protection Agency (EPA), which is the federal agency that develops and enforces environmental laws to protect the environment and human health.

This glossary defines words used by ATSDR in communications with the public. It is not a complete dictionary of environmental health terms. If you have questions or comments, call ATSDR's toll-free telephone number, 1-888-42-ATSDR (1-888-422-8737).

Absorption
The process of taking in. For a person or animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute
Occurring over a short time [compare with **chronic**].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with **intermediate duration exposure** and **chronic exposure**].

Additive effect
A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with **antagonistic effect** and **synergistic effect**].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems.

Aerobic
Requiring oxygen [compare with **anaerobic**].

Ambient
Surrounding (for example, *ambient* air).

Anaerobic
Requiring the absence of oxygen [compare with **aerobic**].

Analyte
A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect
A biologic response to exposure to multiple substances that is **less** than would be expected if the known effects of the individual substances were added together [compare with **additive effect** and **synergistic effect**].

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) **biomedical testing** or (b) the measurement of a substance [an **analyte**], its **metabolite**, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see **exposure investigation**].

Biologic monitoring
Measuring hazardous substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

Biologic uptake
The transfer of substances from the environment to plants, animals, and humans.

Biomedical testing
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

Biota
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

Body burden
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

CAP
See **Community Assistance Panel**.

Cancer
Any one of a group of diseases that occurs when cells in the body become abnormal and grow or multiply out of control.

Cancer risk
A theoretical risk for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Case study
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

CAS registry number
A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.

CERCLA [see **Comprehensive Environmental Response, Compensation, and Liability Act of 1980**]

Chronic
Occurring over a long time (more than 1 year) [compare with **acute**].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with **acute exposure** and **intermediate duration exposure**].

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)
A group of people, from a community and from health and environmental agencies, who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

Comparison value (CV)
Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see **exposure pathway**].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as **Superfund**, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances.

Concentration
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Delayed health effect
A disease or injury that happens as a result of exposures that might have occurred in the past.

Dermal
Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact
Contact with (touching) the skin [see **route of exposure**].

Descriptive epidemiology
The study of the amount and distribution of a disease in a specified population by person, place, and time.

Detection limit
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Disease prevention
Measures used to prevent a disease or reduce its severity.

Disease registry
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

DOD
United States Department of Defense.

DOE
United States Department of Energy.

Dose (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose (for radioactive chemicals)
The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

Dose-response relationship
The relationship between the amount of exposure **[dose]** to a substance and the resulting changes in body function or health (response).

Environmental media
Soil, water, air, **biota** (plants and animals), or any other parts of the environment that can contain contaminants.

Environmental media and transport mechanism
Environmental media include water, air, soil, and **biota** (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The **environmental media and transport mechanism** is the second part of an **exposure pathway**.

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance
The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Epidemiology
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

Exposure
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [**acute exposure**], of intermediate duration, or long-term [**chronic exposure**].

Exposure assessment
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure-dose reconstruction
A method of estimating the amount of people's past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

Exposure pathway
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a **source of contamination** (such as an abandoned business); an **environmental media and transport mechanism** (such as movement through groundwater); a **point of exposure** (such as a private well); a **route of exposure** (eating, drinking, breathing, or touching); and a **receptor population** (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a **completed exposure pathway**.

Exposure registry
A system of ongoing followup of people who have had documented environmental exposures.

Feasibility study
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

Geographic information system (GIS)
A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds
Training sessions for physicians and other health care providers about health topics.

Groundwater
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with **surface water**].

Half-life ($t_{½}$)
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half life is the amount of time necessary for one half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half lives, 25% of the original number of radioactive atoms remain.

Hazard
A source of potential harm from past, current, or future exposures.

Hazardous Substance Release and Health Effects Database (HazDat)
The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Hazardous waste
Potentially harmful substances that have been released or discarded into the environment.

Health consultation
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with **public health assessment**].

Health education
Programs designed with a community to help it know about health risks and how to reduce these risks.

Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or clinical measure and to estimate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over, and to improve, their health.

Health statistics review
The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard
The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence
The number of new cases of disease in a defined population over a specific time period [contrast with **prevalence**].

Ingestion
The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see **route of exposure**].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see **route of exposure**].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with **acute exposure** and **chronic exposure**].

In vitro
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with **in vivo**].

In vivo
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with **in vitro**].

Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual's exposure could negatively affect that person's health.

Metabolism
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of **metabolism**.

mg/kg
Milligram per kilogram.

mg/cm^2
Milligram per square centimeter (of a surface).

mg/m^3
Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see **reference dose**].

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, condition, or injury) is stated.

Mutagen
A substance that causes **mutations** (genetic damage).

Mutation
A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or **NPL)**
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

No apparent public health hazard
A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

NPL [see **National Priorities List for Uncontrolled Hazardous Waste Sites**]

Physiologically based pharmacokinetic model (PBPK model)
A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica
A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see **exposure pathway**].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Potentially responsible party (PRP)
A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb
Parts per billion.

ppm
Parts per million.

Prevalence
The number of existing disease cases in a defined population during a specific time period [contrast with **incidence**].

Prevalence survey
The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

Prevention
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

Public comment period
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

Public availability session
An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

Public health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public health assessment (PHA)
An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with **health consultation**].

Public health hazard
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or **radionuclides** that could result in harmful health effects.

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are **no public health hazard**, **no apparent public health hazard**, **indeterminate public health hazard**, **public health hazard**, and **urgent public health hazard**.

Public health statement
The first chapter of an ATSDR **toxicological profile**. The public health statement is a summary written in words that are easy to understand. The public health statement explains how people might be exposed to a specific substance and describes the known health effects of that substance.

Public meeting
A public forum with community members for communication about a site.

Radioisotope
An unstable or radioactive isotope (form) of an element that can change into another element by giving off radiation.

Radionuclide
Any radioactive isotope (form) of any element.

RCRA [see **Resource Conservation and Recovery Act (1976, 1984)**]

Receptor population
People who could come into contact with hazardous substances [see **exposure pathway**].

Reference dose (RfD)
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see **exposure registry** and **disease registry**].

Remedial investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

Resource Conservation and Recovery Act (1976, 1984) (RCRA)
This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RFA
RCRA Facility Assessment. An assessment required by RCRA to identify potential and actual releases of hazardous chemicals.

RfD
See **reference dose**.

Risk
The probability that something will cause injury or harm.

Risk reduction
Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication
The exchange of information to increase understanding of health risks.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [**inhalation**], eating or drinking [**ingestion**], or contact with the skin [**dermal contact**].

Safety factor [see **uncertainty factor**]

SARA [see **Superfund Amendments and Reauthorization Act**]

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see **population**]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or environment.

Solvent
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an **exposure pathway**.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Stakeholder
A person, group, or community who has an interest in activities at a hazardous waste site.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Substance-specific applied research
A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's **toxicological profiles**. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended CERCLA and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with **groundwater**].

Surveillance [see **epidemiologic surveillance**]

Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see **prevalence survey**].

Synergistic effect
A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see **additive effect** and **antagonistic effect**].

Teratogen
A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Tumor
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a **safety factor**].

Urgent public health hazard
A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Volatile organic compounds (VOCs)
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

Other Glossaries and Dictionaries

Environmental Protection Agency	http://www.epa.gov/OCEPAterms/
National Center for Environmental Health (CDC)	http://www.cdc.gov/nceh/dls/report/glossary.htm
National Library of Medicine (NIH)	http://www.nlm.nih.gov/medlineplus/dictionaries.html

CERTIFICATION

The Health Consultation on *Assessment of Environmental Concerns: Rodney Metals and Brittany Dyeing and Printing Corporation and Evaluation of Cancer Incidence in New Bedford's South End 1982-1998* was prepared by the Massachusetts Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Consultation was initiated.

Gail Godfrey
Technical Project Officer
Superfund Site Assessment Branch (SSAB)
Division of Health Assessment and Consultation (DHAC)
ATSDR

The Division of Health Assessment and Consultation, ATSDR, has reviewed this Health Consultation and concurs with its findings.

for Roberta Erlwein, M.P.H.
Section Chief, SPS, SSAB, DHAC, ATSDR

Assessment of Environmental Concerns: Rodney Metals and Brittany Dyeing and Printing Corporation and Evaluation of Cancer Incidence in New Bedford's South End 1982-1998

Questions and Answers

1. **Q. Why was a study of cancer incidence rates conducted in the South End of New Bedford?**

   A. In response to requests by concerned residents, Representative Antonio Cabral, and the local Health Department, the Community Assessment Program (CAP) of the Massachusetts Department of Public Health (MDPH), Bureau of Environmental Health Assessment (BEHA) conducted an investigation of cancer and environmental factors in New Bedford. The primary environmental concerns were associated with Rodney Metals and Brittany Dye.

2. **Q. How was the study conducted?**

   A. This Health Consultation was prepared by MDPH under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) and followed a standard peer-reviewed protocol for conducting descriptive epidemiologic investigations, approved for use in Massachusetts in 1992. In addition to reviewing descriptive information from the Massachusetts Cancer Registry (MCR) and available environmental data, we considered the most current relevant scientific, medical, and epidemiologic literature regarding chemical toxicity and risk factors for the development of cancer.

3. **Q. What cancer data were used in this study?**

   A. Cancer incidence data for the years 1982–1998 were obtained for the South End area of New Bedford (i.e., census tract 6528) from the Massachusetts Cancer Registry (MCR), a division of the Bureau of Health Statistics, Research, and Evaluation within MDPH. Cancer incidence data consists of reports of newly diagnosed cases of cancer, not reports of cancer deaths. The MCR has been monitoring cancer incidence in Massachusetts by law since 1982. The 17-year period 1982–1998 is the period for which the most recent and complete cancer incidence data were available at the time of this analysis.

4. **Q. What types of cancer were studied and why?**

   A. Eight cancer types were evaluated in this investigation, including cancers of the bladder, breast, kidney, liver, lung, and pancreas as well as leukemia and non-Hodgkin's lymphoma (NHL). These cancer types were selected for evaluation based on elevations that were observed at the city level in a preliminary review of cancer rates in New Bedford and/or to address concerns raised by residents over suspected elevations in some of these cancer types in the South End area.

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5. **Q. Did the study review cancer patterns at the neighborhood level?**

   A. Yes. The census tract (CT) is the smallest geographic area for which cancer rates can be accurately calculated because it is the smallest area for which accurate population counts by age group and gender are available. However, a qualitative evaluation of the patterns of cancer at a smaller geographic level (i.e., the neighborhood level) was also conducted by plotting the address reported for each person diagnosed with cancer on a map to assess any possible concentrations of cases in any one area of New Bedford census tract 6528. (For confidentiality reasons, it is not possible to include maps showing the locations of individuals diagnosed with cancer in the report.)

6. **Q. What environmental data were evaluated?**

   A. To evaluate concerns about potential environmental exposures from Rodney Metals and Brittany Dye, MDPH contacted the Massachusetts Department of Environmental Protection (MDEP) to obtain and review available environmental data for these companies. In addition, we reviewed Toxics Chemical Release Inventory (TRI) data available from the U.S. Environmental Protection Agency (USEPA). An evaluation of potential pathways of exposure was conducted to determine whether releases or activities at the Rodney Metals and Brittany Dye sites could have impacted residents in the South End area. Finally, information regarding other potential environmental sources located in the South End area of New Bedford and listed with MDEP as a location of a hazardous material or oil release (i.e., "21E sites") was reviewed.

7. **Q. Did the study find statistically significantly elevated rates of any type of cancer?**

   A. The majority of the eight cancer types evaluated in New Bedford census tract (CT) 6528 during 1982–1998 occurred approximately at or near expected rates, however, statistically significant elevations were observed in the incidence of leukemia among females during the overall time period 1982–1998 and in the incidence of kidney cancer among males during the most recent time period evaluated, 1995–1998. The rate of lung cancer in this area was statistically significantly lower than expected during 1982–1998.

8. **Q. What did the study find about the geographic pattern of cancer in New Bedford's South End?**

   A. In general, review of the geographic distribution of cancer revealed no apparent spatial concentrations of individuals diagnosed with cancer at the neighborhood level or in relation to the Rodney Metals and Brittany Dye properties or other environmental release sites in this area. However, a concentration of five individuals diagnosed with kidney cancer was observed in close proximity to the Rodney Metals and Brittany Dye facilities. Available data on smoking and occupation were limited, and the possible role of these and other personal risk factors, such as genetics and diet, could not be evaluated for these individuals.

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9. Q. Has kidney cancer been associated with exposure to contaminants of concern at Rodney Metals and Brittany Dye?

A. Contaminants of concern at Rodney Metals and Brittany Dye (e.g., chlorinated volatile organic compounds) have been suggested in some studies to be associated with kidney cancer, liver cancer, and, to a lesser extent, non-Hodgkin's lymphoma (NHL). It is important to note, however, that neither NHL nor liver cancer displayed similar geographic patterns of incidence in this area of New Bedford.

10. Q. If most cancers do not show a pattern related to Rodney Metals or Brittany Dye, does that mean that there are no environmental concerns associated with the facilities?

A. Not necessarily. Environmental contaminants have been detected in subsurface soils and groundwater at Rodney Metals. In addition, both Rodney Metals and Brittany Dye have reported historical air releases of chlorinated volatile organic compounds. It does not appear that area residents are being exposed to subsurface contamination, however, it is not possible to evaluate whether air emissions resulted in the presence of elevated levels of chlorinated volatile organic compounds in the ambient air adjacent to these facilities and, if present, whether human exposure is occurring.

11. Q. Are there non-cancer health concerns associated with these facilities?

A. Residents of the South End area of New Bedford near Rodney Metals and Brittany Dye also expressed concerns about acute non-cancer health outcomes, such as upper respiratory irritation, nausea, and headaches. It is possible that some residents living in close proximity to the two facilities, particularly individuals with pre-existing conditions such as asthma and allergies, could experience some irritant effects associated with chlorinated volatile organic compounds in ambient air. Due to the existence of unpleasant odors and nuisance conditions reported by some individuals residing in areas surrounding the two facilities, MDPH recommends that the Massachusetts Department of Environmental Protection (MDEP) work with both Rodney Metals and Brittany Dye to determine any additional actions that could reduce potential impacts to residents in the surrounding neighborhoods.

12. Q. Does MDPH plan to conduct further study in the South End area of New Bedford?

A. Yes. Based on the observed geographic concentration of kidney cancer diagnoses in proximity to the Rodney Metals and Brittany Dye facilities during the most recent time period evaluated, 1995–1998, MDPH will provide additional follow-up for all individuals diagnosed with this cancer type in New Bedford census tract (CT) 6528 during 1982–1998. Specifically, the 12 individuals (or their families) who provide informed consent will have the opportunity for personal interviews and/or medical records review by an environmental/occupational physician to determine any possible environmental or other factors that may have contributed to their kidney cancer diagnosis.

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13. Q. Where can I obtain a copy of the Health Consultation *Assessment of Environmental Concerns: Rodney Metals and Brittany Dyeing and Printing Corporation and Evaluation of Cancer Incidence in New Bedford's South End, 1982-1998*?

A. The full report is available on the MDPH, BEHA web site at www.state.ma.us/dph/beha. In addition, a copy of the report is available at the New Bedford Free Public Library (Main Library).

14. Q. Who should I contact for more information?

A. For more information on the Health Consultation *Assessment of Environmental Concerns: Rodney Metals and Brittany Dyeing and Printing Corporation and Evaluation of Cancer Incidence in New Bedford's South End, 1982-1998*, please contact:

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www.state.ma.us/dph/beha

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